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FRIEDBERGER AND FRÖHNER'S  
VETERINARY PATHOLOGY

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[Authorised Translation]

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VOLUME I



# Friedberger & Fröhner's Veterinary Pathology

[Authorised Translation]

TRANSLATED AND EDITED BY

M. H. HAYES, F.R.C.V.S.

*Author of "Points of the Horse," "Veterinary Notes for Horse Owners," etc.*

WITH NOTES ON BACTERIOLOGY

BY

DR. G. NEWMAN, D.P.H.

VOLUME I


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## PREFACE TO THE 4TH EDITION.

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SINCE the publication of the last edition of this manual, so many new discoveries and researches have been made in the domain of epizootic pathology, that it has been necessary to considerably enlarge the contents of this book. In order to avoid too great an increase in its size, it was necessary to make several omissions, as for instance, the introductory diagnostic chapters. These deleted paragraphs will be found in our text-book on the methods of clinical research. We have tried to gain space for new additions, by introducing abbreviations and by altering the type. The additions in question had to be ended by the 1st October, 1895.

In our last edition we have mentioned the French and Russian translations of this book. Since then, an unauthorised English translation of that edition has been brought out in America by a Dr. Zuill.

FRIEDBERGER.  
FRÖHNER.

MUNICH AND BERLIN.

*February, 1896.*



## TRANSLATOR'S PREFACE.

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FRIEDBERGER and Fröhner's *Lehrbuch der speciellen Pathologie und Therapie der Haustiere*, of which this book is an authorised translation, is not only the highest authority on veterinary medicine in Germany, but its French translation by Cadiot and Ries has been adopted as a text-book on that subject by the French veterinary colleges. Owing to the fact that the infective diseases of animals can in many cases be communicated to man, and that their occurrence in our meat and milk producers is a serious question of public hygiene; the first volume of this translation will appeal to doctors as well as to veterinary surgeons. The second volume will more particularly concern the veterinary profession.

In the *Addenda to Infective Diseases* (pp. 455—483), I have ventured to supply the omission, in the original, of articles on Surra, South African Horse Sickness, Australian Tick Fever, Ixodic Anæmia in Jamaica, and Louping-ill, and have added a few remarks on *The Terms Infection and Contagium* (pp. 511—512) with the object of throwing light on a subject upon which there is a good deal of confusion of ideas. The remarks made within square brackets and foot-notes have been added with the view of meeting the requirements of English readers.

I am greatly indebted to Professor Friedberger for the kind assistance he has given me in explaining the meaning of certain words and phrases which occur in the original, and which I should have been unable to have translated without his aid. I have also to thank Mr. H. Lang (co-author of Treves and Lang's *German-English Medical Dictionary*) and Mr. Otto Ruppelius for help in this translation.

I am particularly obliged to Dr. G. Newman, M.D., D.P.H., Medical Officer of Health, for his *Notes on Bacteriology* (pp. 484—510), which I feel certain will greatly aid those readers who are not very familiar with that science, in understanding the bacteriological portion of this book.

I beg gratefully to thank Dr. Lingard and Professor Meek for their respective help with the sections on Surra and Louping-ill; Professor McFadyean for notes on Contagious Pneumonia of the Pig, and Swine Fever; Mr. Alexander Cope, Chief Inspector, Veterinary Department of the Board of Agriculture, for assisting me in compiling statistics of animal diseases in Great Britain; Mr. Sidney Villar for his notes on pages 85 and 89; and Mr. Wilcox for his note on page 448.

I may state that I purchased the right of translating this book from the German publisher, Mr. Ferdinand Enke, previous to undertaking this translation.

As the metric weights, measures, and degrees of temperature have been retained in this translation, tables for converting them respectively into those which are generally used in this country, are given on pages xi. and xii.

In the present edition of this translation, Dr. Newman has revised and enlarged his chapter on bacteriology, which appeared in the previous edition; and I have adopted a similar course with respect to the notes I made, and to the chapter on *Addenda to Infective Diseases*.

M. H. HAYES.

YEW TREE HOUSE, CRICK, RUGBY.

21st May, 1904.



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## WEIGHTS, MEASURES AND DEGREES.

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As the metric system, which has been adopted by Continental scientific men, is not generally used by veterinary surgeons in English speaking countries ; the following tables are given by the translator. Readers of this book should bear in mind, that when the word, gramme, is employed in connection with fluids, it means a cubic centimetre (c.c.); because that measure of pure water at 4° C (39.2° F.), weighs a gramme. In prescriptions, when numbers are given without any allusion to weights or measures, grammes are meant.

### RELATION OF METRIC WEIGHTS TO ENGLISH WEIGHTS.

1 Milligramme	=	0.015432 grains.
1 Centigramme	=	0.15432 „
1 Decigramme	=	1.5432 „
1 Gramme	=	15.432 „
1 Kilogramme	=	2 lbs. 3 oz. 120 grains (nearly).

### RELATION OF METRIC FLUID MEASURES TO ENGLISH FLUID MEASURES.

1 Cubic centimetre (c.c.)	=	17 minims (nearly).
1 Litre	=	1.76 pint (nearly).

### RELATION OF METRIC MEASURES OF LENGTH TO ENGLISH MEASURES OF LENGTH.

1 Millimetre	=	0.03937 inches.
1 Centimetre	=	0.3937 „
1 Decimetre	=	3.937 „
1 Metre	=	39.37 „

### RELATION OF CENTIGRADE DEGREES TO FAHRENHEIT DEGREES.

A difference of  $1^{\circ}$  C. is equal to that of  $1.8^{\circ}$  F.; but their respective zeros are not the same. For instance, the freezing point of water is represented by  $0^{\circ}$  C., according to the metric system; and by  $32^{\circ}$  F., according to the popular English idea. Hence, to convert centigrade degrees into Fahrenheit degrees, we have to multiply them by 1.8 and add 32. For instance, if we wish to find how many Fahrenheit degrees are in, say,  $40^{\circ}$  C., we can do so, by the following equation:

$$40^{\circ} \text{ C.} = 40 \times 1.8^{\circ} \text{ F.} + 32^{\circ} \text{ F.} = 104^{\circ} \text{ F.}$$

If the required conversion is that of  $-20^{\circ}$  C., the equation would be

$$-20^{\circ} \text{ C.} = -20 \times 1.8^{\circ} \text{ F.} + 32^{\circ} \text{ F.} = -4^{\circ} \text{ F.}$$



## CHAPTER I.

### INFECTIVE DISEASES.

SEPTICÆMIA AND PYÆMIA—MALIGNANT ŒDEMA—PETECHIAL FEVER—STRANGLES—DISTEMPER—MALIGNANT CATARRHAL FEVER—ENZOOTIC ABORTION—DYSENTERY—ERYSIPELATOUS DISEASES OF PIGS—SWINE ERYSIPELAS—CONTAGIOUS PNEUMONIA OF THE PIG AND SWINE FEVER—CHOLERA AND PLAGUE OF BIRDS—DEER AND CATTLE DISEASE—QUARTER-ILL—INFLUENZA—EQUINE CONTAGIOUS PLEURO-PNEUMONIA—TUBERCULOSIS—ACTINOMYCOSIS—STOMATITIS PUSTULOSA CONTAGIOSA OF HORSES—DIPHThERITIC DISEASES—PARASITIC STOMATITIS—TETANUS—GLANDERS—BOVINE PLEURO-PNEUMONIA—DOURINE—VESICULAR EXANTHEMA OF HORSES—VESICULAR EXANTHEMA OF CATTLE—ANTHRAX—RABIES—FOOT AND MOUTH DISEASE—VARIOLA—RINDERPEST.

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### SEPTICÆMIA AND PYÆMIA.

Septicæmia and pyæmia in general—Septicæmia of fowl—Septicæmia of geese.

**General Remarks.**—The term, septicæmia, is applied to infective diseases which are characterised anatomically by tumefaction of the spleen and cloudy swelling of the liver, kidneys, heart, and other internal organs. As these anatomical changes are met with in other diseases, as for instance in anthrax, swine-erysipelas, chicken-cholera, "deer and cattle disease" (p. 106), quarter-ill, etc.; such diseases must also be put under the heading of septicæmia. The foregoing infective diseases are simply specific varieties of septicæmia, that is to say, forms of septicæmia which have distinctive names and well-recognised contagia. Besides the specific forms of septicæmia, we have simple septicæmiæ which have no distinctive names applied to them, and which usually take their starting-point from wounds; although they may also become developed in the internal organs,

especially in the intestines and lungs. In surgery they are classed generally as blood-poisoning.

Septicæmia is clinically distinguished from pyæmia by the fact that no metastatic suppurating foci are formed in the internal organs of animals suffering from it. Etiologically, there exists no essential difference between the two, which are frequently so intimately combined together that a strict differentiation between them is not always possible. In such cases, we speak of a septicopyæmia. Strangles is a specific pyæmia.

**Causes of Septicæmia.**—The two chief causes are : (1) bacteria which produce “septic infection” ; and (2) dissolved chemical poisons (toxins) which give rise to “septic poisoning.” Septic infection, but not septic poisoning, can be transmitted to other animals by inoculation of affected blood. There are various transitions and combinations between the two forms.

1. *Bacterial septicæmia* [septic infection] is caused by various kinds of bacteria (cocci and bacilli). Consequently, septicæmia which is originated by cocci is different from that produced by bacilli. Streptococcus septicus and micrococcus tetragenus are the chief septicæmia-producing cocci. The cocci of pus, namely, streptococcus pyogenes and staphylococcus pyogenes aureus, can also give rise to septicæmia. Koch proved experimentally that the respective bacilli of the septicæmia of mice and of the septicæmia of rabbits are pathogenic, as are also those of enteritis, bacteria coli, and the bacilli of malignant œdema.

2. *Septic intoxication* consists in the absorption of poisonous products of the metabolism of bacteria, such as tox-albumens, organic bases, fatty acids, and aromatic products. The absorption of these poisons usually takes place from a serous focus which is on the surface of the body or in some internal organ, such as the intestines, lungs, and liver, and which is occupied by the before-mentioned pathogenic bacteria. Non-pathogenic fungi, especially the bacteria of putrefaction, can produce very strong chemical poisons by the septic decomposition of the animal tissues, after these fungi have penetrated into the purulent or serous foci. The absorption of these products of metabolism gives rise to general intoxication. This form of septicæmia, which is caused by putrefactive fungi (saprophytes), is called “sapræmia.” Proteus vulgaris, proteus mirabilis, and proteus Zenkeri are the most dangerous kinds of these fungi.

**Causes of Pyæmia.**—The micro-organisms of pyæmia are

on the whole the same as those of septicæmia, and may be either cocci or bacilli. The most frequent exciters of pyæmia are the pus cocci, especially streptococcus pyogenes and staphylococcus pyogenes aureus, which give rise to metastases by emigrating from a primary suppurative focus into the blood vessels. They are then carried by the blood-stream to various internal organs, where, on becoming arrested in the capillaries, they set up suppuration. Pyæmia most frequently occurs from purulent breaking down of thrombi of veins in wounds, mucous membranes (as in puerperal uterus), lungs, hoofs, and navel; injuries of bones; and absorption of pus in internal purulent foci, as for instance, the suppurating lymph glands in strangles and pharyngitis. Pyæmia may also occur after contagious equine pleuro-pneumonia.

**Anatomy.**—The most important appearance on *post mortem* examination of a case of septicæmia, is the changes of the blood, which assumes the colour of lacquer, coagulates imperfectly and swarms with bacteria. We find cloudy swelling and degeneration of the muscular tissue of the heart, liver, kidneys, and muscles of the skeleton, which often look like boiled meat; frequently, swelling of the spleen and hæmorrhages in the mucous membranes of the head, intestinal canal, and bladder; hæmorrhages in the lungs, liver, kidneys, and muscles, and under the endocardium, etc. Septic endocarditis and enteritis sometimes appear. We may also observe an accumulation of colonies of septic bacteria in the kidneys (nephritis bacteritica) and other organs. The cadaver decomposes very rapidly and in some cases forms great quantities of fœtid gas. The walls of the capillaries are thickly studded with bacteria, and their lumen is often filled up with masses of fungi. The leucocytes frequently contain bacteria. When the course of the disease has been very acute, the anatomical changes are often so slight that they may be easily overlooked.

In cases of pyæmia, besides the pus cocci in the blood, we find abscesses and formation of pus in various organs (pyæmia metastatica), chiefly in the lungs, liver, kidneys, and muscles. Morot found, for instance, more than a hundred abscesses in the muscular system of a cow which was suffering from pyæmia. We meet with suppuration of the serous membranes—such as the peritoneum, pleuræ, meninges, and synovial membranes of the articulations—and of the eye, in the form, for instance, of purulent choroiditis and panophthalmia. We may observe

ulcerous endocarditis and numerous circumscribed hæmorrhages in the serous membranes, skin, eyes, muscles, etc. There may also be anatomical changes peculiar to septicæmia (septicopyæmia).

**Symptoms.**—The first symptom in septicæmia is generally that of high fever, with a temperature of  $42^{\circ}$  C. or more, and sometimes preceded by rigors. The action of the heart is very feeble, and the pulse frequent, small, and finally imperceptible. In some cases the temperature is normal. This preliminary stage is soon followed by general disturbance, great debility, dulness of the mental faculties, muscular tremors, occasionally paralysis of the hind-quarters, dirty-red or icteric discoloration and ecchymoses of the mucous membranes, discoloration and albuminous condition of the urine, and complete loss of appetite. Continued diarrhœa appears towards the end. As a rule, local affections are absent. The disease generally lasts for several days, and may continue for some weeks; or it may terminate fatally in a few hours. Recoveries are rare.

The metastatic appearance of pus in various organs distinguishes pyæmia from septicæmia, which presents only general symptoms. The pyæmic changes are accompanied by a very irregular fever, which is often intermittent, and by rigors. According to the localisation, we find metastatic inflammation of the lungs, which usually ends in gangrene of the lungs; abscesses in the liver, kidneys and brain; and multiple subcutaneous deposits of pus, which often appear quite suddenly and sometimes in great numbers, in the form of phlegmonous swellings on different parts of the body; pleuritis; meningitis; polyarthritis; and suppurating inflammation of the spinal cord, with paralysis of the hind-quarters. Pyæmia sometimes gives rise to a hæmorrhagic diathesis with hæmorrhages in the internal organs and mucous membranes. Its course is longer than that of septicæmia, and varies from several days to several weeks, according to the seat and extent of the lesions. The mortality is not so great as in septicæmia; although, even in pyæmia, recoveries are rare, and the period of convalescence is always long. The course of the disease is very erratic, on account of the complications which may occur in various organs.

**Diagnosis.**—A diagnosis of non-traumatic, ordinary septicæmia is often very difficult; for the chief symptoms to be relied on are high fever and constitutional disturbance,



which may be present in other diseases, such as anthrax. In pyæmia, we have also metastatic or embolic suppuration in various parts of the body, with, usually, a primary suppurating focus. Acute glanders is the disease for which it may be most readily mistaken.

**Therapeutics.**—The treatment of septicæmia consists in the internal administration of antiseptics, the most appropriate being camphor, quinine, and calomel in repeated small doses. Camphor may be used externally in the form of linimentum camphoræ, or spiritus camphoræ; and its stimulant action is valuable in combatting the excessive weakness. The various modern antipyretics may also be tried. The antiseptic treatment of the wound plays the principal part in traumatic septicæmia. In pyæmia, which is almost always incurable, the same remedies are used as in septicæmia. The treatment is essentially surgical.

**Septicæmia of Fowl.**—Legrain and Jacquot have described a form of pyæmia which is peculiar to fowl, and which frequently occurs in Lorraine, where it causes great loss. It is characterised by extensive abscesses on the head and neck, and especially about the eyes. If these abscesses are left alone, they may produce a chronic septicæmia, which leads to severe and progressive emaciation that ends in death; but if treated surgically, they rapidly heal. The disease can be transmitted to pigeons (which die in from 15 to 30 hours) and mice, and is caused by short, stout bacilli which have rounded and shining ends. These bacilli are from 1 to 2  $\mu$  long, and cannot be stained by Gram's method. Their cultures, according to Legrain and Jacquot, resemble those of the bacillus septicus agrigenus of Nicolaïer, the bacillus saprogenus of Rosenbach, and the bacillus pyogenes foetidus of Passet, which these authors consider to be identical with each other. The poultry infect themselves on the dunghills in which they seek their food.

**Septicæmia of Geese.**—Sakharoff describes a typhus-like summer epidemic of Russian geese, which manifests itself by high fever, diarrhœa, and emaciation, and runs a fatal course in about a week. He found in the blood of living animals, spirochæta anserina, which resemble the spirilla of relapsing fever, and which he successfully transmitted to geese.

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#### MALIGNANT ŒDEMA.

**Etiology.**—Koch's malignant œdema, which Pasteur termed "gangrenous septicæmia," is of special clinical importance. It is caused by small spore-forming bacilli (the bacilli of œdema,

the vibrios septiques of Pasteur) which are ubiquitous bacteria, and which occur mostly on the surface of the ground. A rabbit will die in from 24 to 36 hours from malignant œdema if a small quantity of ordinary garden-soil is placed under its skin. The infection takes place in consequence of contamination of wounds of the skin by earth, fæces, hay-dust, etc. ; but only when the bacillus of œdema gets into the sub-cutaneous connective tissue. The introduction into the blood of such infective material is comparatively innocuous ; apparently because the oxygen of the blood kills these anærobes. Inoculation from the cutis is also unsuccessful. The process of granulation hinders the entrance of the bacilli into wounds. Before the bacilli of œdema can penetrate into it, the sub-cutaneous connective tissue must be, to a certain extent, prepared for their reception, as for instance by the presence of such nutrient fluids as serum, lymph, and blood. The site of the infection must also be as free as possible from blood which contains oxygen. Consequently, the more the circulation is interrupted on the site of the infection, the better will these bacilli develop on it. Chauveau states that an attack confers subsequent immunity.

The bacilli of œdema closely resemble in size and shape those of anthrax, except that they are more slender, are under certain circumstances motile, and have rounded ends. After the death of infected animals they become greatly elongated, and form partly stiff, partly slightly-bent and loop-like threads on which divisions can be recognised, so that they present the appearance of bacilli attached to one another. Later on, the spores develop from these threads. After 24 hours, the bacilli of œdema are found in great numbers in the blood (especially in that of the portal vein) of animals which have died from suffocation, if the cadaver has been kept at a temperature of 38° C. For that reason they may be found, as a rule, within from 12 to 24 hours in the blood of the liver, and afterwards throughout the entire blood of the body in the cadavers of large domestic animals which have died of dyspnœa, colic, etc., and which retain their internal temperature for a long time. Lustig believes in the possibility of horses becoming infected through the intestines by the bacilli of œdema, and describes cases in horses to support this theory.

According to the investigations of Kitt, malignant œdema may be easily transmitted by inoculation to all our domestic animals, including guinea-pigs, fowl, and pigeons.

**Bacteriology.**—The bacilli of malignant œdema are spore-forming rods, anaerobic and generally very motile. They liquefy gelatine, can be stained with Gram's solution, are from 3 to 5  $\mu$  long, and 1  $\mu$  broad, and are consequently about four times as long as they are broad. Several rods usually form psuedo-threads of from 10 to 40  $\mu$  in length. Although the bacilli are at once destroyed by oxygen, the spores resist it and most disinfectants. During growth, the bacilli cause the production of carbonic acid, hydrogen, sulphuretted hydrogen, and carburetted hydrogen, which gases are the cause of emphysema. In the connective tissue, the bacilli increase only in places from which blood and air are excluded as much as possible, and assume in it the form of spore-bearing or homogeneous rods. On the serous membranes the bacilli become greatly elongated, and afterwards undergo division. Only towards the end of the disease, or after death, do they penetrate into the blood, where they are then found in the form of short rods, or as simple micrococci. Their vitality is arrested by putrefaction, and altogether ceases in about two months' time. If the bacilli are dried at a temperature of from 15° to 38° C., before putrefaction sets in, they will permanently retain their virulence. They show a considerable power of resistance to antiseptics. When fresh, they are killed by a temperature of 100° C. in 15 minutes; and when dried, by one of 120° C. in 10 minutes. Cornevin, Chamberland, and Roux obtained positive results with experiments in rendering animals immune against this disease.

**Anatomical and Clinical Notes.**—The principal changes in malignant œdema consist in an œdematous, doughy and painful swelling in the neighbourhood of the infected part. This swelling spreads more and more, and is often found to crackle on palpation. In the centre, it is generally relaxed, cool, and painless; but tense, hot and painful on its periphery. The subcutaneous connective tissue, adipose tissue, and neighbouring muscles are infiltrated with yellow gelatinous material, and contain foetid gas bubbles. The orange-coloured fluid of the œdematous swelling contains numerous characteristic bacilli of œdema and threads, which are absent in the blood during life, and are found after death only in small numbers. Besides, we find inflammation of the mucous membrane of the small intestine and œdema of the lungs. The serous fluid in the alveoli of the lungs contains great quantities of bacilli and threads. The spleen, liver, and kidneys are unaffected, and it is noteworthy that the spleen is not swollen. The disease usually runs a fatal course in from 24 to 48 hours, with very severe fever. When only a few of the bacilli of œdema are inoculated, recovery with local formation of abscesses may occur.

**Differential Diagnosis.**—Kitt draws attention to the

great similarity between malignant œdema and quarter-ill. The spread of the latter is, however, restricted within enzootic limits; but the bacilli of the former are ubiquitous and show a characteristic thread formation. Quarter-ill is generally fatal. We distinguish malignant œdema from anthrax by the fact that the bacilli of the former are not present in the blood of living animals; are occasionally motile; have rounded ends; and that cutaneous inoculations with their cultivations in mice and guinea-pigs give negative results. Besides, the appearance of the respective cultures are essentially different. See also p. 338.

**Therapeutics.**—The treatment of malignant œdema is chiefly surgical, and consists in making incisions into the emphysematous parts so as to admit air, the oxygen of which is the best agent for destroying the bacilli of œdema; in obtaining drainage; and in freely disinfecting the skin.

Kitt surmises that the bacilli of malignant œdema may be the cause of many diseases which are at present classed under the respective headings of septicæmia, bovine erysipelas, and the often inexplicable œdema of the lungs of cattle. It is certain that some cases of septic puerperal fever in cattle and sheep, and probably many so-called phlegmonous swellings, are simply malignant œdema, which appears to be the cause of the œdematous and generally fatal swellings sometimes seen after phlebotomy, rowelling, bites of dogs in sheep, bites in dogs, castration, wounds of the tongue in horses from extraction of teeth, and operation wounds made most likely with dirty instruments. Friis reports a case of malignant œdema which appeared in a horse after an injection of eserine. Attinger, Reuter, Elmenhoff, and Horne described several cases in cattle.

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#### PETECHIAL FEVER.

(*Horse typhus, Morbus maculosus, Purpura hæmorrhagica.*)

Petechial fever in horses—Typhus of man—Petechial fever in cattle.

**Former Views on Petechial Fever.**—This disease was formerly known as typhus or typhoid fever on account of its supposed identity with these diseases. Hering called it "petechial fever" after its most important symptom, viz., that of hæmorrhages in the mucous membranes; although he laid particular stress on the fact that petechiæ on the mucous

membranes also occur, as a mere symptom, in diseases, such as strangles, which have no connection with petechial fever.

In England, petechial fever was regarded by some as scarlatina; by others, as morbus maculosus of man (purpura hæmorrhagica). The name of morbus maculosus was commonly applied to it in Germany, as for instance by Eberhard. In France and Italy it was looked upon by some authorities, and is regarded even at the present time by Trasbot, as acute anasarca; and was traced, not to decomposition of the blood, but to great and temporary paralysis of the capillaries and effusion of serum and blood. Lafosse and others stated that it was either septicæmia or anthrax. Owing to the teaching of Röhl and others, petechial fever was supposed for a long time in Germany to be an anthracoid disease, which theory was erroneous; because it is neither infectious nor can it be transferred by inoculation to horses or to other animals. Besides, the bacilli of anthrax have never been found in the blood of horses suffering from it; and some of its symptoms are incompatible with the supposition of anthrax. Arloing failed to transmit it, even by transfusions of blood.

Dieckerhoff considered that petechial fever was similar to morbus maculosus Werlhofii of man, and proposed that the name, "morbus maculosus," which was already in use, should be retained. We prefer the designation of petechial fever, and leave undecided the question of its relation to morbus maculosus, the causes and nature of which are not yet clearly known. We agree, however, with Dieckerhoff that the names of "horse typhus" and "petechial typhus" should not be employed; for petechial fever has nothing in common with the typhus of man.

**Etiology and Pathogenesis.**—Petechial fever is an acute infective disease, the cause of which is unknown. It is characterised by numerous hæmorrhages in the skin, subcutis, mucous membranes, submucosa, and internal organs, with subsequent inflammatory oedematous swellings of the skin, mucous membranes, subcutis, and submucosa. Although the disease may appear primarily; it is usually a sequela to previous infective diseases, such as strangles, pharyngitis, contagious equine pleuro-pneumonia, and influenza, and, chiefly, during convalescence after these diseases. Chronic catarrh of the lungs, chronic inflammation of the maxillary sinuses, necrosis of bone, intestinal catarrh with suppuration

of the lymph follicles, abscesses in the lungs, kidneys, and spleen, and infected wounds, such as those after castration, have been observed to be the starting-points of this disease. It sometimes follows a slight injury to the skin. We do not know whether the virus is a chemical or a bacterial product.

Zschokke has observed in dépôts of cavalry horses, the occurrence of several cases in one and the same stable.

We are as ignorant of the origin of the contagium, as of its nature, and know nothing about the manner in which it enters the body. As horses which are used in the cleaning out of latrines, are especially liable to be attacked by this disease; we may surmise that the infection is conveyed in the respired air.

A disease identical with petechial fever of horses appears to occur in cattle, especially in cases of inflammation of the uterus, mastitis, etc. We have several times noticed in dogs, hæmorrhagic affections of the character of human morbus maculosus; but no ailment which corresponded to the petechial fever of horses.

**Bacteriology.**—Kolb recently found, 3 or 4 hours after death, in people who had suffered from morbus maculosus (purpura hæmorrhagica), a bacillus to which he gave the name of "*bacillus hæmorrhagicus*." This bacillus forms oval, thick, non-motile rods with rounded ends, in the form of diplo-bacilli, attached to one another, and sometimes forming pseudo-threads. They cannot be stained with Gram's solution. Pigeons and guinea-pigs were found to be immune; but dogs, rabbits, and mice became affected with a hæmorrhagic ailment. The bacilli were seen in great numbers in the blood of the cadaver. Letzerich states that he has discovered the specific bacillus of morbus maculosus. These statements require confirmation.

**Anatomy.**—The anatomical changes consist in hæmorrhages in the skin, subcutis, mucosa and submucosa of the nasal cavity, larynx, trachea, brônchi, conjunctiva, pharynx, oral cavity, stomach, intestine, bladder and vagina, lungs, spleen, kidneys, and muscles of the heart and skeleton. Zschokke assumes as the cause of these hæmorrhages, a mycotic endarteritis with the formation of thrombi and subsequent infarcts. These changes, as in poisoning by phosphorus, may be due to fatty degeneration, and brittleness of the arterial walls in consequence of injury caused by the virus. The smallest hæmorrhages are found in the nasal mucous membrane, where they sometimes vary in size from a millet seed to a lentil. The largest are in the lungs, where they may attain the size

of a man's fist. As a consequence of the hæmorrhage, we find inflammatory sero-gelatinous swellings in the subcutis, submucosa, and inter-muscular tissue, which may lead even to sloughing of the affected parts, especially of the skin of the flexor surfaces of the joints, intestinal mucous membrane, and less frequently of the mucous membrane of the nose and pharynx. Hæmorrhages into the cavities of the body and even death may occur from laceration of hæmorrhagically infiltrated parts, or a parenchymatous inflammation may become developed, as for instance, in the lungs. Owing to gangrene of the inflamed tissues; pyæmia, septicæmia, or perforative intestinal peritonitis may set in. The individual organs show the following changes :—

1. The *skin*, especially on the most depending parts of the body, as on the legs, muzzle, brisket and abdomen, becomes locally or diffusely swollen, and infiltrated with hæmorrhages, which vary in size from a pea up to half-a-crown, and which give the skin a spotted appearance after it has been removed from the body, *post mortem*. The skin over the swollen parts frequently shows lacerations and sores which may penetrate into the muscles. The subcutaneous cellular tissue appears on section to be infiltrated with serous fluid and blood, and is also considerably swollen.

2. The *mucous membrane of the nasal cavity* is covered with irregularly shaped blood spots which vary in size from a lentil up to a hazel nut, and which frequently become confluent and thus form large sanguineous blotches. We sometimes find the entire nasal mucous membrane infiltrated to such an extent with blood, that the lumen of the nasal cavities is considerably narrowed. The infiltration is usually greatest in the mucous membrane of the turbinate bones. The nasal mucous membrane may show ulcers which may penetrate so deeply, as to produce holes in the septum nasi. Similar infiltrations and hæmorrhages are found in the mucous membrane of the nasal cavity and larynx, with consequent œdema of the glottis. The epiglottis and the guttural pouches are the seat of a sanguineous infiltration, and ulcers appear in the pharynx as well as in the larynx. The retro-pharyngeal lymph glands frequently become swollen and suppurate. Petechiæ are not very numerous in the oral cavity, in which they are chiefly found on the gums, tongue, and inner surface of the swollen lips, and may run into ulcers. The conjunctiva and the vaginal mucous membrane sometimes become swollen and covered with petechiæ. The

mucous membrane of the stomach shows red spots. Hæmorrhages occur under, as well as in, the mucous membrane, under the serosa and in the muscular layers, in which they are generally so finely divided, that the muscular tissue, on section, has a greyish-blue colour. In the intestinal canal we find masses of exudate which vary in size from a lentil to the palm of one's hand, and which have been thrown off by the mucous membrane, submucosa, and subserosa. The muscular coat of the intestine is infiltrated with minute hæmorrhages. The mucous membrane is more or less swollen and is frequently the seat of ulcers, which vary in size from a quarter of an inch to four inches in diameter. These ulcers, which have a tinder-like base, sometimes granulate and form scars. At other times, they perforate the walls of the intestine and set up peritonitis. The contents of the intestine are sometimes mixed with blood. The changes are best marked in the small intestine; although they are also found in the cæcum, colon and even in the rectum, in which parts the hæmorrhages often take the form of streaks.

3. *The muscles of the skeleton*, such as those of the pelvis, thighs, abdomen, chest, tongue, the masticatory muscles, etc., frequently show great numbers of dark-brown or copper-coloured localised hæmorrhagic infiltrations of various forms and sizes. The muscular fibres often become granulated and undergo fatty degeneration, so that the affected muscle becomes pale and of a clay colour. The interspersed hæmorrhages may give a marbled appearance to the muscles. The intermuscular connective tissue is frequently gelatinously and hæmorrhagically infiltrated. Portions of muscular tissue sometimes become gangrenous and are cast off. The muscular tissue of the heart becomes similarly affected; it may contain numerous sub-epicardial, subendocardial, and interstitial hæmorrhages; and may assume a similar clay or greyish-red colour. There may also be hæmorrhages under the periosteum.

4. The *lungs* are infiltrated with miliary, subpleural, and parenchymatous blood foci, which may vary in size from a walnut to a man's fist, and by running together, may produce a diffuse hæmorrhage of the lungs. We also observe extensive croupy, or multiple suppurating and gangrenous pneumo ic patches which are due to foreign bodies, hypostasis of the lungs, and œdema of the lungs.

5. We sometimes find in the *spleen* and *kidneys*, large nodular hæmorrhages, the rupture of which may cause death,



by the entrance of blood into the abdominal cavity. There is no distinctive swelling of the spleen. The cavities of the body often contain moderate quantities of bloody transudate; and the abdominal cavity occasionally contains a large amount of fluid blood.

6. The *blood* shows no essential changes, as long as the disease progresses without complications. No bacteria have as yet been found during life; although the bacilli of malignant œdema can be demonstrated in it soon after death, as well as in horses which have died from suffocation.

7. Besides the above-mentioned phenomena, we find secondary changes resembling those of *septicæmia* or *pyæmia*, and of *suffocation*; and at times, the residual changes of the primary affection (strangles for instance) which preceded the petechial fever.

**Symptoms.**—The first symptom is usually the appearance of numerous dark red *petechiæ*, *i.e.*, blood foci, on the more or less congested nasal mucous membrane. They are sometimes as small as a flea-bite; but are generally from one quarter to three quarters of an inch in diameter, and frequently run together so as to form large blotches or streaks. Owing to their presence, the nasal mucous membrane assumes a peculiar dotted or speckled appearance, and may become purple throughout its entire extent, if the petechiæ be very numerous. It is at the same time more or less swollen, and exudes serous fluid. In very severe cases the mucous membrane becomes gangrenous, and covered with ulcers; the nasal discharge is frequently sanious and discoloured; and the expired air has a fœtid odour. In such cases, respiration becomes difficult, and the disease may take a rapidly fatal course with severe aggravation of the general symptoms. The eruption of the petechiæ may continue during the entire course of the disease.

Simultaneously with, or a few days after, the appearance of petechiæ on the nasal mucous membrane, we find *cutaneous swellings* which are often the first symptom observed, on account of their being more readily seen than the blood spots. These swellings take the form of wheals, and are from three quarters to one and a half inches in diameter. They may be distributed over the whole body, but are generally found on the most dependent parts, such as the limbs, lower portion of the face, abdomen, sheath, and lower surface of the chest. These swellings at first vary in size from an apple to a man's fist, and resemble

carbuncles. Later on they become confluent or spread upwards, so that we have sharply defined diffuse swellings, which greatly disfigure the attacked parts. The extremities increase in circumference; large tumours form on the lower surface of the chest and abdomen; and the lower part of the face becomes like that of a hippopotamus. The swellings are hard and painful to the touch, and when they occur on the head they are sometimes as hard as a board. The hair on them often falls out. The nose-band of the halter frequently makes an indentation in the swelling on the nose. The rapid and simultaneous appearance of swellings on several parts of the body is characteristic of this disease. When the swelling is severe, lacerations of the affected skin of the flexor surfaces of the articulations are very liable to occur. Cutaneous gangrene may supervene, with consequent sloughing of pieces of skin, and the formation in their place of indolent ulcers. The smooth, dense skin, first of all exudes a few drops of sanious serum, after which it becomes cold and insensible to the touch, and exfoliates off in pieces the size of a bean up to that of the palm of a man's hand; leaving a sore which may extend to the underlying muscles. Apart from this spontaneous, anæmic, cutaneous necrosis, the skin has a great tendency to form sores from continued lying down, and from other kinds of pressure, especially on the nose (where the nose-band comes), "girth place," edges of the lower jaw, bones, shoulders and orbital arches. The rapid disappearance of the cutaneous swellings is sometimes an unfavourable sign which may mean even death, when it is due to a sudden and abundant sero-hæmorrhagic extravasation in the intestinal mucosa with subsequent absorption of the subcutaneous infiltration; or when it is caused by a rapid rise of temperature, in which case the increased metabolism leads to the absorption of any nutrient material that may be present.

Swellings on the *limbs* render the gait awkward, prevent the animal from lying down and getting up, give rise to expressions of pain when the limbs are being bent, and often make it impossible for the horse to be moved from the spot he occupies. When the lower part of the head is considerably swollen, the opening of the nostrils becomes proportionately narrowed, and sometimes even completely closed, with the result of excessive dyspnœa, a wheezing form of breathing, and finally suffocation, unless the condition is relieved in time.

As regards the *digestive apparatus*, we have to point out that

the mucous membrane of the oral cavity sometimes shows petechiæ, but rarely ulcers. At the commencement of the attack, food may be eaten fairly well for several days ; although petechiæ may be present on the nasal mucous membrane, and swellings on the integumentum commune. Later on, mastication often becomes difficult and finally impossible by swelling of the lips and cheeks. With the appearance of fever, the appetite becomes disturbed and constipation sets in. Swallowing is frequently prevented by grave implication of the pharynx, on the mucous membrane of which, petechiæ and œdematous swelling of the submucosa appear with the formation of ulcers and inflammation of the retro-pharyngeal lymph glands.

In such cases, the petechial fever is complicated with symptoms of *pharyngitis*, such as difficulty in swallowing, salivation, regurgitation, wedging-in of food between the teeth and walls of the cheeks, continued extension of the head and the neck, bad smell from the mouth, and swellings on the neck. The patient generally becomes rapidly worse. A very unfavourable complication is the appearance of colicky pains, which are usually of an intermittent nature, and are caused by hæmorrhages in, and subsequent inflammatory swelling of, the intestinal mucous membrane, and in some cases by intussusception of the intestine. In the course of these attacks of colic, the lumps of dung that are passed are often covered with muco-purulent membranes, which fact points to the presence of inflammation of the mucous membrane of the rectum. We may observe, on protrusion of the anus during defecation, that the mucous membrane of the rectum is swollen and infiltrated with hæmorrhages. Such colics may rapidly lead to death in consequence of paralysis or perforation of the intestines.

The urine frequently contains considerable quantities of blood (*hæmaturia*). Micturition may be rendered difficult by swelling of the opening of the sheath. Petechiæ may be sometimes seen on the mucous membrane of the vagina.

At the beginning of the disease, the *temperature of the body* is usually only slightly, if at all, raised. In rare cases, the disease is ushered in by a temperature of from 40° to 41° C. The petechiæ are in no way connected with the fever, which is usually moderate, and averages from 39.5° to 40° C. Higher degrees of temperature are observed chiefly when complications occur. The frequency of the pulse is not increased at the commencement of the attack ; although

it becomes strikingly high, as soon as painful inflammatory swellings appear on the skin. Its rate is then, as a rule, from sixty to eighty per minute; but increases on the appearance of complications. Even in normal cases, the increased frequency of the pulse is out of all proportion greater than the rise of temperature. As a rule, the mental faculties are not greatly deadened; although, later on, if the disease assumes a fatal course, stupor and excessive weakness set in. The animal frequently suffers from copious outbreaks of sweat.

Apart from the swelling of the nostrils, *respiration* becomes disturbed only when there is excessive inflammation of the laryngeal mucous membrane or inflammation of the lungs. In the former case, we notice sounds due to stenosis of the larynx, which may cause death by suffocation in consequence of œdema of the glottis. The lung trouble is often overlooked, because its symptoms are not well marked. Sometimes hæmorrhage of the lungs may be diagnosed by the fact of the expectoration being mixed with blood; a croupy inflammation of the lungs, by the increased area of dulness combined with bronchial breathing; gangrene of the lungs, by the fœtid smell of the expired air, and grave general disturbances; and œdema of the lungs, by the rapidly increasing dyspnœa combined with crackling râles, which can be heard by auscultation.

The *eyes* are sometimes affected and the conjunctiva often becomes infiltrated with hæmorrhages, and may even become so swollen that it prolapses. The lachrymal fluid may be sanious or orange red. By means of the ophthalmoscope, we have been able to find in the interior of the eye, iris, choroid and retina, hæmorrhages which had taken place independently of external influences. Röhl has observed destruction of the eyes from excessive hæmorrhage; and Schindelka, atrophy of the optic nerve from the same cause.

**Course.**—The course of petechial fever is very atypical and irregular. In some mild cases, absorption of the hæmorrhagic and inflammatory foci takes place even within a week, without any grave complications; although in the majority of the mild attacks, the formation of hæmorrhagic infiltrations and swellings of the skin lasts from 8 to 14 days, after which, improvement gradually sets in. If, however, the hæmorrhagic infiltration of the skin and the mucous membranes is extensive, especially if the mucous membrane of the

pharynx and intestines becomes involved, absorption of the inflammatory transudates will not usually take place earlier than in from 4 to 6 weeks, or even later. Cases have occurred in which the swellings of the skin and mucous membranes disappeared quite unexpectedly and without treatment. These cases of sudden improvement have been observed even in animals which have been given up by the veterinary surgeon who treated them. Zschokke found, among 17 cases, that the disease lasted, on an average, 16 days. The shortest duration, which occurred in a fatal case, was 40 hours; the longest, 43 days. Death may ensue in a few days, in consequence of internal hæmorrhages, rapid sepsis, suffocation, paralysis of the small intestine, or when the attack has been the result of a grave primary disease. Gangrenous pneumonia (caused by foreign bodies) is particularly fatal. If death takes place after the petechial fever has lasted for a considerable time, its cause will generally be secondary septicæmia or pyæmia, the symptoms of which will be: debility, high internal temperature, depression and diarrhœa. Ulcerating skin lesions require, as a rule, several months to heal. They may also cause cicatricial contractions, especially when they occur on the flexor surface of joints, producing, for instance, excessive uprightness of the pasterns. In such cases it is almost always better to kill, than to put the animal under a long course of treatment; for prognosis is very unfavourable.

**Differential Diagnosis.**—The respective course may run simultaneously with petechiæ on the nasal mucous membrane, and with circumscribed swellings of the skin (wheals). In horses, these diseases are chiefly strangles, anthrax, septicæmia, contagious pleuro-pneumonia, pyæmia, acute glanders, and erysipelas; and in cattle, quarter-ill, "deer and cattle disease," septicæmia puerperalis, other forms of septicæmia, malignant œdema, and poisoning by phosphorus and mercury. Consequently, it is not always easy to differentiate these diseases from petechial fever, especially at the commencement of an attack. The presence of diffuse swellings of the skin with extensive eruption of petechiæ, and the absence of the specific bacilli of anthrax, quarter-ill, malignant œdema, etc., will make the diagnosis positive in most cases. The same remark holds good in the differentiation of petechial fever from urticaria and simple cutaneous cellulitis.

**Prognosis.**—As the disease is liable to many complications and as its course is generally very tedious, our prognosis should be given with great caution. The mortality averages 50 per cent. In 17 cases observed by Zschokke 12 deaths occurred (70 per cent.). The following are unfavourable symptoms: extensive formation of petechiæ and inflammatory swellings; tendency to necrosis of the skin and mucous membranes; grave implications of the mucous membrane of the pharynx, larynx, and intestines; stenosis of the nasal cavities by swellings; high internal temperature; frequent pulse, exceeding 80 per minute; entire loss of appetite; rapid disappearance of the swellings of the skin; fœtid smell of the expired air; inflammation of the lungs; excessive weakness; inclination to decubitus; profuse diarrhœa; and great depression. We may take a favourable view of those cases in which the bodily condition is well sustained, the extent of the hæmorrhages and swellings slight, the appetite good, and the internal temperature not much above normal. As we have already stated, severe symptoms are sometimes followed by unexpected amelioration and recovery. We should therefore not be too hasty in ordering the slaughter of horses which appear to be very gravely affected.

**Therapeutics.**—With regard to hygiene, our first care should be to provide a roomy loose-box in which the animal can freely move about; and we should remove the head-stall and surcingle. When difficulty in swallowing sets in, we should, if possible, give green food, or, failing that, mashes. Among the many medical agents which have been used to combat this disease, we may mention calomel, salicylic acid, sugar of lead, tannic acid, ergot of rye, extractum hydrastis, preparations of iron, camphor, quinine, arsenic, carbolic acid, iodide of potassium, creolin, lysol, ichtyol, oil of turpentine, sulphuric acid, and hydrochloric acid. Some give boric acid (20 grammes in the drinking water) or cinchona bark (50 grammes as an electuary). All these medicines produce at times a good effect; but in the large majority of cases they fail to favourably influence the disease.

We may try to relieve the nasal dyspnœa in the first place by passing a suture through the median alæ nasi, drawing it up and laying it over the middle line of the nose. Or we may follow Johne's advice and introduce metal tubes into the nostrils. In most cases, however, when this

dyspnoea is complicated with laryngeal stenosis, tracheotomy will be our only available resource. We do not agree with the idea held by some that this operation should be performed earlier than necessity demands, so as to obviate the risk of the wound becoming gangrenous; because gangrene may supervene as a consequence of the accidental occurrence of septicæmia. Local treatment of the cutaneous swellings with astringents also appears to us to be usually superfluous. In particular, we have often applied Burow's solution (alum, 1 part; acetate of lead, 2 parts; water, 100 parts); but with varying results. We use aluminium acetate as an external antiseptic only when the swellings show breaches of continuity. In all other cases, we employ only inunctions of pure oil or paraffin ointment to lessen the tension. Inunctions that irritate or cause inflammation are decidedly objectionable. Like Röhl, we have seldom found any advantage, but rather the contrary, from scarifications of the most swollen parts of the skin, with the object of obviating sloughing. On the other hand, Dieckerhoff reports favourably on the effect of deep incisions of the swellings. The treatment of the sloughing and ulcerating parts of the skin is purely surgical. Aruch recommends cold irrigations continued for several days as a good remedy for excessive swelling of the head.

**The Treatment of Petechial Fever with Iodine.**—Dieckerhoff has strongly recommended intratracheal injections once daily of 10 to 30 grammes of Lugol's solution (iodine, 1 part; potassium iodide, 5 parts; and distilled water 100 to 200 parts). The numerous reports in the literature of the last few years, on the effect of these injections of iodine are very contradictory; in fact, while some praise them highly, others speak of them as absolutely inefficient, and even as injurious. Zschokke, who treated more than a dozen cases of petechial fever with injections of iodine, considers that their results were not so good as those which he had obtained with the remedial means he had formerly employed. Johne states that all the 3 horses which were treated with injections of iodine in Dresden died; out of 2 others which were treated with iodine and calomel at the same time, 1 died; and 1 horse which was treated exclusively with calomel recovered. In two cases, injections of iodine were followed by necrotic tracheitis and gangrene of the lungs. We ourselves have observed the occurrence of granular tracheitis and bronchitis as a result of these injections. Lemke and Buch report that horses died of 30 grammes of the above-mentioned solution of iodine. As about 50 per cent. of the cases of petechial fever recover without any medicinal treatment, and as apparently hopeless cases of this disease sometimes recover without treatment; we cannot view with favour the iodine method.

**Typhus of Man.**—Three forms of typhus are distinguished in man,

namely, abdominal typhus, or ileo-typhus; the exanthematic typhus, or petechial typhus; and relapsing typhus, or relapsing fever.

1. *Abdominal typhus* is produced by a specific bacillus and is a miasmatic contagious disease, which essentially consists in a necrotic infiltration of the follicular lymph glands of the small intestine, especially of the ileum near the ileo-cæcal valve. There is considerable swelling of the spleen and catarrh of various mucous membranes. It begins with rigors and considerable stupor, followed by typical fever; persistent diarrhœa; bronchitis; severe nervous disturbances, such as delirium, twitching of the tendons, and the appearance of red spots (three to six) on the chest or abdomen. We find very varying complications, such as internal hæmorrhages, perforation of the intestines, angina, ulcers on the larynx, parotitis, severe inflammation of the lungs, nephritis, affection of the brain, embolus, paralysis, etc. The mortality now-a-days is only about 7 per cent. All experiments made with feeding animals even on the excrements of people suffering from typhus, have given negative results. Baumgarten proved by his experiments that the human bacillus of typhus could not be transmitted to rabbits, guinea-pigs, or mice. Semmer is the only veterinary writer who speaks of the occurrence of human typhus in domestic animals (horse and dog).

2. *Petechial typhus of man* is highly infectious, and in certain districts is an endemic infective disease, as, for instance, in the form of "famine fever." On *post mortem* examination, no characteristic changes are found; only purely general changes. In particular, the intestinal ailment, which is characteristic of abdominal typhus, is absent. During the course of the disease, which is very typical, the skin becomes affected with a roseola exanthema, that develops later on into petechiæ, namely, true hæmorrhages. Improvement and recovery often take place with surprising rapidity. The mortality is only about 10 per cent.

3. *Relapsing fever* is caused by spirilla, namely, the spirochæta Obermeyer, which are found in the blood. It runs its course without producing any special organic changes. Its chief symptom is very high fever, the course of which is marked by exacerbations and remissions. The mortality is only 2 per cent. Human relapsing fever does not occur in animals.

**Petechial Fever in Cattle.**—The symptoms are, as a rule, similar to those already described, and are as follows:—Petechiæ on the mucous membrane of the nose, eyes, and vagina; epistaxis; bloody diarrhœa; hæmaturia; petechiæ and extensive hæmorrhagic swellings in the skin, especially on the extremities, dewlap, and hypogastrium; lameness; paralysis; high temperature (up to 42° C.); loss of appetite; and suppression of rumination. In a few cases, dyspnoea and abnormal sounds from stenosis of the air passages, in consequence of hæmorrhagic swellings of the respiratory mucous membranes, have been observed. The aspect of the disease may remind one of anthrax, quarter-ill, "deer and cattle disease," malignant œdema, septicæmia, or mercurial poisoning. When the petechiæ appear on the mucous membrane of the mouth, the disease may be mistaken for aphthæ epizooticæ. *Post mortem* examination reveals hæmorrhages and hæmorrhagic swellings in the skin, subcutis, mucous membranes of the organs of digestion and respiration, serous membranes, heart, brain, kidneys, etc. The course



of the disease appears, on the whole, to be more rapid and unfavourable in cattle than in horses.

The multiple hæmorrhages of the muscular system, which often occur in pigs, are not petechial fever ; but, according to Ostertag, lacerations of the fibres of the muscles caused during the transport of the animals. Ellinger assumes, in addition to this, an acquired hæmorrhagic diathesis as a predisposing cause.

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## STRANGLES.

Strangles in horses—Pseudo-strangles of dogs.

**Etiology.**—Strangles is an acute infective equine disease which consists essentially of an infectious catarrh of the mucous membrane of the upper air passages, especially of the nasal cavity, with suppuration of their lymph glands. According to Schütz, Jensen, and Sand, the infective agent is a streptococcus, which can be easily found in cover-glass preparations of the pus of the lymph glands, after having been stained with aniline dyes, such as gentian violet. It can also be stained with Gram's solution. Rabe states that the mother-cells of the streptococci of strangles are ovoid bacilli, which, when connected together, sometimes assume a square form, and possess polymorphous properties. These bacilli may take the form of threads, or slender rods which remind us of the bacilli of glanders ; or they may be oval or even circular, and may sometimes form tetrads. In broth, they form a flocky mass at the bottom of the vessel, and on solid mutton serum, glassy, transparent drops, which later on become confluent, in which case cloudy masses are seen in each drop in the serum test-tubes. They have a pathogenic effect on white mice, and produce suppuration in the site of the inoculation, and metastatic processes by means of the lymphatics and blood vessels. When pure cultivations of the streptococci of strangles are inoculated in horses, they produce abscesses in the site of the inoculation ; and strangles, when they are introduced into the nasal cavity.

**Pathogenesis.**—Strangles is contagious, and, perhaps, also miasmatically contagious. Schütz states that the cocci of strangles form arthro-spores, namely, permanent cells, which probably exist, and which may also grow under specially favourable circumstances, outside the animal body. Successful ex-

periments in the transmission of strangles have been made many years ago, as for instance, by Lafosse, in 1790; Viborg, in 1802; Erdelyi, in 1813; and by Reynal, Toggia, and others. The contagium is found principally in the nasal mucus, and becomes disseminated in the air. Even in the last century, Erasmus Darwin assumed that a miasma which is distributed in the air is the exciting cause of strangles.

Strangles is peculiar to horses, asses, and their hybrids. It appears sporadically, enzootically, and epizootically, and most frequently attacks horses of from 2 to 5 years of age. Older horses, even up to 25 years of age, as was shown by Jensen, Sand, and others, may also suffer; and also foals during the first few weeks or months after birth. According to the observations of Nocard, Wiart, Sourdrel, Choisy, and Frederik, strangles may be transmitted *in utero*. In such cases, the streptococci pass from the mother to the foetus, the internal organs of which will then contain numerous suppurating foci with the characteristic streptococcus of strangles. Similar cases have been observed in former times. According to Jonsson, strangles is unknown in Iceland, which fact can be easily explained by the contagious character of the disease. One attack confers immunity for certainly a couple of years, and frequently for the remainder of the horse's life.

Youth is the chief predisposing cause; for susceptibility to the disease is inversely proportionate to the age of the animal; although, as before mentioned, it is never absolutely effaced. As other predisposing causes, we may mention: weakening of the vital powers by chill; catarrhal affections of the mucous membrane; bad climatic conditions, especially change of weather in spring and autumn; pampering; continued confinement in the stable; over-exertion; transport over long distances; and an irregular system of stable management. The disease spreads epizootically, particularly when large numbers of horses are kept together, as in cavalry depôts, in the army, in the stables of horse-dealers, and in breeding establishments.

The contagium is absorbed chiefly through the respiratory mucous membrane. The high initial internal temperature, which is manifested even prior to the swelling of the glands, and the experimental observations of Schütz, Jensen, and Sand, prove that the infective matter may be taken into the blood directly from the mucous membrane. In its further progress, the contagium keeps to the direction of the lymph passages and

enters by preference into the lymph glands, in which it excites extensive suppuration. As was shown by Schütz, the cocci of strangles can also pass through the walls of the capillaries without the help of the blood or of the lymph, and can penetrate between the tissue cells of the affected organs. It is probable that the contagium may enter the body also from the intestinal mucous membrane. This supposition is strengthened by the fact of the occurrence of abscesses in the mesenteric glands, with a normal condition of the nasal mucous membrane and the simultaneous absence of abscesses in the submaxillary gland; by the existence of abscesses in the submucosa of the intestinal mucous membrane; and by changes in Peyer's patches and the solitary glands, which can sometimes be demonstrated. The same remark applies to the lungs, the lymph glands of which, namely, the bronchial glands, are often found to be in a suppurating condition.

The period of incubation appears to vary from 4 to 8 days.

**History.**—The former views on the nature of strangles are now only of historical value. This disease was regarded as due to chill or development, or was connected with a supposed lymphatic equine idiosyncrasy. Dentition and a peculiar state of the weather were also regarded as causes of it. Others considered strangles to be identical with scrofula or with human measles. In more recent times, Trasbot, as Sacco had previously assumed, declared that strangles were true horse-pox. Trasbot, as Viborg and Toggia had done in their time, proposed that all horses should be inoculated with the lymph of horse-pox as a prophylactic measure against strangles. This hypothesis, which is in itself quite improbable, has been refuted experimentally by Delamotte, who demonstrated that the inoculation of horse-pox does not protect horses against strangles. Furthermore, the fact of the existence of different forms of strangles was advanced; as for instance, strangles of foals; benign, suspicious, malignant, slow, metastatic, occult, gangrenous, putrid, asthenic, compound, and vesicular strangles; strangles caused by infectious matter which had to be eliminated from the body; strangles originating from glanders; etc.

**Symptoms of ordinary, mild Strangles.**—An attack of strangles begins by a rise of internal temperature varying from  $40^{\circ}$  to  $41^{\circ}$  C. on an average. During the following days, the temperature falls  $0.5^{\circ}$  to  $1^{\circ}$  C., and does not again rise until suppuration takes place in the lymph glands. It decreases simultaneously with the outward discharge of pus. At the commencement of the disease, the pulse usually remains more or less normal in spite of the high temperature, and only later

on, its rate increases to 50 or 60 beats a minute. A higher frequency is met with only in animals of very weak constitution and when complications arise.

The first local symptom consists in catarrh of the nasal mucous membrane, which generally becomes diffusely congested; although, at times, the redness may be confined to certain spots. The discharge is at first serous and viscid; but after about three days it becomes mucilaginous and finally purulent, and then its colour varies from a dirty-white to a yellowish green. This purulent catarrh is usually found in both nasal cavities; but may, in exceptional cases, be more or less unilateral. In young animals, it is generally very abundant; but in aged horses it is sometimes very slight.

In the large majority of cases, swelling of the submaxillary glands appears concurrently with the purulent nasal catarrh. This swelling is hot and painful to the touch. The spreading of the inflammation to the connective tissue which surrounds the glands, and the stasis of the lymph in the efferent lymph vessels, often cause the development, from the submaxillary glands, of very extensive swellings that may occupy the entire intermaxillary space, and may spread even to the exterior side of the posterior maxilla. The course of the swelling of the submaxillary glands may vary. In by far the greater number of cases, abscesses form; but we may sometimes have dispersion and absorption with gradual resolution or induration. The formation of an abscess becomes known by the appearance, in the swelling, of soft spots, which at first are indolent, but later on are distinctly fluctuating. The skin over these spots is tense, purple in colour, and covered with a lymph-like viscid exudate. The hair over these spots falls out; the skin covering them becomes thin, discoloured and necrotic; and the abscess "points," spontaneously bursts, and discharges from one or more openings a thick, yellowish-white, creamy pus, which is sometimes mixed with necrotic shreds of tissue. The abscess cavity heals by granulation. As soon as the abscess bursts, which it will generally do in from 8 to 12 days, if it has not been previously opened, the swelling will rapidly disappear from the part, and the internal temperature will fall as quickly as it rose. In exceptional cases, strangles may present only catarrhal symptoms without suppuration of the lymph glands. Jensen states that it may first assume the form of pharyngitis, purulent pneumonia, and pleuritis, without any well-marked morbid affection of the lymph glands.

Among accompanying symptoms we may note: loss of appetite, which is partly due to the general feverish affection, and partly to the swelling of the intermaxillary space; depression; occasionally, excessive weakness at the beginning of the attack; œdematous swellings, especially of the hind-legs; and often great emaciation, if the disease has lasted a considerable time. In spite of the very high temperature, the urine generally remains alkaline; although it frequently contains a considerable quantity of albumen. During convalescence, we may sometimes observe a temporary polyuria, as in other acute infective diseases.

At times, strangles is accompanied by a cutaneous exanthema which usually takes the form of an eruption of wheals, the size of a pea up to that of half-a-crown, and spreads over the entire body; or an eruption of nodules, vesicles, and even pustules, chiefly on the lateral surface of the neck, shoulders and sides of the chest. These exanthemata are characterised by their sudden appearance, and often by their equally rapid disappearance. An eruption of vesicles may break out on the nasal mucous membrane, and in exceptional cases may spread to the skin of the *alæ nasi* and upper lip. The contents of the vesicles are at first limpid, and become purulent later on. The vesicles soon burst, and leave shallow ulcers, which become covered with a thin scab and heal without leaving a scar. These vesicular processes on the nasal mucous membrane give rise to the idea that they are characteristic of a peculiar kind of strangles, for which, some writers have evidently mistaken cases of stomatitis pustulosa contagiosa, and frequently those of inflammation of the lymphatics. Elevations on the nasal mucous membrane resembling wheals are rare. They take the form of circumscribed, dark-red, raised, sero-hæmorrhagic infiltrations of the mucous membranes. They vary in size from a lentil up to a pea, and are often surrounded by a red-coloured edge. Rabe states that the streptococci of strangles can produce ulcers on the nasal mucous membrane in consequence of croupy and diphtheritic infiltration of the mucous membrane (see remarks on the differential diagnosis of glanders, p. 282).

**Complications of Strangles.**—I. One of the most frequent complications is the spreading of the infectious catarrh to the *pharyngeal mucous membrane*, with the consequent production of *pharyngitis*, which, in a few cases, may be the first symptom of strangles. Pharyngitis, thus transferred, is well marked. The patient suffers from difficulty in swallowing,

salivation, regurgitation, and swelling and suppuration of the retro-pharyngeal lymph glands (the so-called upper cervical glands or subparotideal glands). The affection of these glands manifests itself outwardly by a diffuse and often very extensive swelling in the lower parotid region, above the larynx; and the lymph glands near the temporo-maxillary articulation directly under the ear, and the lymph glands lying between the lobules of the parotid gland, may also participate in the inflammation. These glandular swellings usually terminate in suppuration and discharge of pus, partly outwards and partly inwards in the direction of the pharynx and larynx. In other cases the abscess undergoes caseation and becomes partly absorbed, or becomes fluid with subsequent septicæmia or pyæmia. The abscesses may attain an extraordinary size by several small ones becoming confluent, and the connecting tissue surrounding the pharynx often becomes densely infiltrated with suppurating foci. Smaller abscesses occur under the pharyngeal mucous membrane and in the muscular apparatus of the pharynx. A phlegmonous abscess in the subcutis may become developed from the retro-pharyngeal lymph glands, and may give rise to extensive swellings of the skin of the head and neck. Under the most favourable circumstances, the pus in the suppurating lymph glands works outwards, either directly or after burrowing a way for itself. Or it may burst inwards into the pharyngeal cavity by reason of the mucous membrane having become necrotic on account of pressure exercised by the abscess,\* in which case the production of gangrenous pneumonia is a frequent sequence. The abscess may open inwards as well as outwards, and thus cause a pharyngeal fistula. Fistulæ may form in a similar way from the submaxillary glands, and may in a few cases open into the oral cavity.

2. The infectious inflammation may spread from the nasal cavities to the *mucous membrane of the larynx, trachea, bronchi, accessory cavities of the nose, oral cavity, eyes, and guttural pouches*. A superficial catarrhal inflammation, the irritation from which sets up cough, becomes developed at first in the larynx, and, sometimes, on spreading downwards, becomes phlegmonous, and may thus lead to severe dyspnoea. In the course of this *laryngitis* we may have the formation of submucous abscesses, inflam-

\* I venture to submit that the local destruction of tissue is due, not to pressure, but to the action of the ferment or ferments which are formed by the pus-bacteria, and which have the property of dissolving albumen.—TR.

mation and paralysis of the muscles of the larynx, and roaring, or "whistling." The catarrh of the bronchi may lead to swelling and suppuration of the bronchial glands. Masses of fodder are, in a few cases, found wedged in the guttural pouches in consequence of necrosis of its walls, and may penetrate even into the subcutaneous tissue along the throat around the trachea. In strangles the guttural pouches, are however, rarely implicated.

3. Inflammation of the *superficial lymph vessels of the skin of the head*, with the formation of a large number of minute abscesses, is chiefly met with on the cheeks, lips, *alæ nasi*, and sometimes over the entire head as far as the ears, even down to the throat. The numerous lymph vessels of the implicated skin are then inflamed, and the connective tissue which surrounds them becomes infiltrated with pus. This perilymphatic suppuration produces in the course of the lymph vessels, numerous minute abscesses which spring from rosary-like nodules that have nothing to do with the suppuration of the lymph glands. This inflammation of the lymph vessels may be followed by diffuse, phlegmonous swelling of the lips and cheeks, in which case the mucous membranes of these parts become tense and frequently as hard as a board. There may also be simultaneous suppuration of the mucous glands which, on the eversion of the tensely swollen lips, will appear as plugs of pus, and will give rise to minute ulcers.

4. Strangles is sometimes characterised by its tendency to form *metastatic abscesses* in a great variety of organs (pyæmia of strangles), partly from the neighbouring lymph glands by means of the lymph passages, and partly by embolism through the agency of the blood. Thus we find abscesses with peripheral swelling in the neighbourhood of the thyroid gland directly in front of the trachea; middle cervical glands behind the trachea (with lateral swelling on the throat in the middle of the jugular groove, which swelling may sometimes burst on both sides and may cause roaring); omo-hyoideus and sterno-hyoideus muscles; lower cervical glands (with formation in front of the entrance of the trachea into the thoracic cavity, of a tumour which may cause compression of the trachea, and suffocation or permanent stenosis of the trachea); axillary glands (producing lameness); subcutaneous tissue, especially on the lateral thoracic walls, front of the chest, lower ribs, hypogastrium, flanks, scrotum, udder, neck, withers, inner surface of the thighs, etc. These subcutaneous abscesses are accompanied by extensive swelling of the skin of the affected parts, the condition of which is fre-

quently mistaken for mere œdematous swellings of the skin. Besides the swelling and suppuration of the bronchial glands, we find in the thoracic cavity metastatic mediastinal abscesses which vary in size from a pea to a man's head, and which produce by their rupture a purulent pleuritis (empyema). Similar abscesses, which often contain as much as 10 litres of pus, and which give rise to fatal peritonitis by their perforation into the abdominal cavity, may be found in the mesentery, omentum, pelvic connective tissue, mesenteric glands, pancreas, kidneys, etc. These abscesses may lead in some cases to adhesions between the intestines. Their presence is manifested during life by periodic and often chronically recurrent slight attacks of colic, and may in a few cases be ascertained by exploration *viâ* the rectum. Abscesses which are accompanied by swelling and suppuration of the intestinal follicles, and which are also characterised by frequently occurring remittent colic, are sometimes found under the mucous membrane of the stomach and intestines. Numerous abscesses of greater or less size and purulent infiltration may be observed in the brain (with symptoms of inflammation of the brain, chronic hydrocephalus, blindness, and local symptoms); spinal cord (paraplegia of the hind limbs, and paralysis of the rectum and bladder); joints, especially in the knee, hock, fetlock, and coronary articulation (with suppurating synovitis, swelling, and functional disturbances); udder (suppurating mastitis); orbit (with consecutive suppurating pachymeningitis); and lymph glands of the extremities, particularly in the popliteal glands, abscesses in which may cause lameness that may last for weeks, and emaciation of the muscles of the hip. When the popliteal glands are thus affected, the swelling will become visible only at a late period on account of the deep position of the glands under the gastrocnemii muscles. If the abscess is not opened early, it will burrow for itself a passage towards the inner surface of the thigh. Animals which have been thus affected sometimes continue permanently lame after recovery from strangles.

5. Complications may arise in the lungs in the form of suppurating (metastatic) *pneumonia* due to foreign bodies and simple catarrhal pneumonia. Generally speaking, pneumonia caused by foreign bodies in cases of pharyngitis, is set up by the entrance of the pus of pharyngeal abscesses into the air passages. Contagious equine pleuro-pneumonia may also be a complication of strangles. Jensen states that the streptococci of strangles may be found in the pyæmic pulmonary abscesses



which occur during the course of contagious equine pleuropneumonia.

6. Strangles assumes a chronic lingering course, when a *chronic catarrh*, which keeps up the nasal discharge, becomes established in the sinuses of the head, and in the guttural pouches, or in the pharyngeal cavity. The animal becomes very thin and suffers from indigestion, and frequently appears as if it was affected with glanders (the so-called suspicious strangles), in consequence of the similarity of these symptoms with those of chronic glanders.

7. Death from strangles is usually caused by the additional influence of *septicæmia* or *pyæmia*. In such cases, there is high fever and great depression; the pulse is frequent and weak; palpitation of the heart ensues; the animal becomes debilitated, often in a very rapid manner; and severe diarrhoea sometimes sets in towards the end.

8. Strangles is sometimes complicated with *petechial fever*, most frequently during the stage of convalescence.

**Differential Diagnosis.**—Strangles is mistaken chiefly for simple nasal catarrh which is accompanied by a purulent nasal discharge, and which is tolerably frequent, particularly in young animals. It is distinguished from strangles by the fact that it never leads to suppurating inflammation of the submaxillary glands, although in a few cases it may cause swelling of these glands. To distinguish strangles from glanders, see the differential diagnosis of glanders, p. 282, *et seq.* In doubtful cases it is advisable to inoculate white mice, which are immune from glanders; although they are typically susceptible to the inoculation of strangles. Strangles may also be mistaken for parotitis and mucous degeneration of the turbinate bones.

**Prognosis** is favourable; because the mortality is small. Thus, out of 5,520 remounts and horses in the ranks of the Prussian army which suffered from strangles in the years 1888 to 1893, only 2 per cent. died. In the Bavarian army during the last two years there was a mortality of 2.5 per cent. Ringsheim states that among 2,205 horses of the Danish army which suffered from strangles, only 0.5 per cent. died during the years 1851 to 1860. This surprisingly low percentage may be explained by the fact that the ages of these horses were from 4 to 9 years. Krabbe on one occasion found among 1,789

affected horses of every age, a mortality of 1.6 per cent. ; and at another time among 1,107 sick horses, one of 3.5 per cent. During 1885, in Denmark, Goldschmidt observed a mortality of 2.3 per cent. among 2,419 affected horses. During 1883, the mortality in Denmark was 2.6 per cent. among 2,381 affected horses. We may consequently assume that the average mortality of strangles is about 3 per cent. Among unfavourable conditions we may mention : debility from previous illness, youth, and complications, especially metastatic pyæmia.

**Therapeutics.**—In normal cases, it is sufficient to attend to hygiene, which consists in cleanliness, assiduous ventilation of the stable, and the administration of food which can be easily digested, such as “green meat,” mash,es, and roots. It is useless to give febrifuges to reduce the initial high internal temperature, which will abate of itself under normal conditions in a few days. The treatment of the suppurating lymph glands is purely surgical ; its essential principle being to open the abscesses with a long incision as early as possible, in fact, as soon as pus forms in them. The early discharge of the pus is quickly followed by a fall of the internal temperature, and evidently shortens the duration of the disease ; consequently we may reject the old custom of allowing the abscesses to “ripen” in a natural manner. If the swelling is indolent, we may stimulate the formation of pus by fomentations or blisters.\* We should open the abscess with caution on account of the proximity of numerous blood vessels, and should divide only the skin with the knife, and then work up to the abscess with the finger. Failing to penetrate the abscess, it is often necessary to make a second attempt in another direction in the event of our not wishing to explore with a trocar and cannula.

For catarrh of the mucous membranes in strangles, practitioners generally use inhalations of steam and internal remedies, such as preparations of antimony (crude sulphide of antimony and orange), and alkalies (sulphate of soda, chloride of ammonium, bicarbonate of soda) combined with liquorice, honey, etc. If the catarrh proves obstinate, we may give in each feed a teaspoonful of the officinal artificial

\* I have had most excellent results, by strongly blistering the swelling, as soon as it appears, with biniodide of mercury ointment (1 to 4 of lard). I am inclined to think that, in these cases, the good effect of the blister is due to the high antiseptic action of the biniodide of mercury, which, according to Cadéac, is 34 times greater than that of corrosive sublimate.—TR.

Karlsbad salt, or an electuary of sulphate of soda 250 grammes, crude sulphide of antimony 25 grammes, with liquorice powder and althæa powder, twice a day.

Complications must be treated according to their nature. Tracheotomy has sometimes to be performed to relieve excessive dyspnœa. For cases in which pharyngitis supervenes, Kagel recommends the introduction of a strong indiarubber tube of about 60 cm. in length into the nostrils as far as the upper end of the nasal cavity, with subsequent injection of about 50 c.c. of a disinfecting solution. From our own experience we can speak favourably of this local treatment. The separation of healthy animals from those which are affected, is necessary for prophylaxis.

**Inoculation.**—The experiments made by Jensen and Sand with intravenous injections of the coccus of strangles, produced no general infection, but only violent cellulitis, which, however, conferred immunity from subsequent infection through the nasal mucous membrane. We have to wait for further experiments with this method before giving a decisive opinion on it. It is certain that in Germany, Peterson's very peculiar method of producing infection will not find many disciples. He exposed all the foals of a breeding establishment during autumn to severe cold, by driving them into a pond and keeping them there for half-an-hour, and then took them to a hill, where they were exposed on all sides to cold winds. After that, he gave them very cold water to drink. All these animals became affected with strangles, but recovered completely in 3 weeks.

**Pseudo-strangles of Dogs.**—Puppies may suffer from a purulent follicular inflammation of the lips with purulent lymphangitis and purulent lymphadenitis of the head, which may lead to pyæmia, and which consequently possesses a certain similarity to strangles. As it is an original infective disease which is independent of distemper and pyæmia, we may give it the name of "canine strangles."

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## DISTEMPER.

Distemper of dogs—Distemper of cats.

**History.**—Canine distemper has always been regarded as a very dangerous disease to dogs. Laosson states that it was known in the time of Aristotle, and considers that it was the canine epizootic which raged in Bohemia during 1028. It is now the general opinion that distemper was imported to Europe for the first time about the middle of the 18th century from

America (Peru, according to Heusinger). It was first brought to Spain, from which it was taken to France, Germany, and other countries. It is said to have reached France about the year 1740; Germany about 1748; Italy about 1764; England about 1760; and Russia about 1770. At the present time it is spread all over Europe.

Formerly distemper was regarded as plague, nervous febris mucosa, typhoid, and even typhus identical with that of man. Many old authors considered it to be true small-pox, with which opinion Trasbot agrees; and numerous unsuccessful experiments were made with vaccine as a preventive against it. This hypothesis has been refuted by the recent experiments of Dupuis, who in no instance was able to produce immunity against distemper by vaccinating young dogs. Many supposed it to be a disease of development or a constitutional affection.

It was recognised by Waldinger, von Gemmeren, Delabère Blain and others at an early period, that distemper was spread by an infective agent; and consequently numerous artificial transmission experiments were made with its virus; the first successful experiments being those by Renner and Karle. Trastowo proved by his experiments, that young dogs which had not suffered from the disease were susceptible to it; that it could be transmitted directly and indirectly; and that old dogs could be infected by it. Trasbot was at first unsuccessful with his inoculation experiments; but later on he was able to transmit it by inoculation to dogs, from 13 days to 3½ months old, by introducing the nasal discharge mixed with the contents of the vesicle, into small incisions on the abdominal walls, with the result that the first symptoms of the disease appeared in 8 days. He also produced infection through cohabitation, which experiment was confirmed by Venuta, who further proved that the contagium was fixed as well as volatile, and that it had sufficient vitality to bear drying in the air up to a certain degree. Venuta found that the period of incubation varied from 4 to 6 days. Krajewski inoculated 36 dogs, of which the greater number remained unaffected; because, as he surmises, they had passed through a previous attack of distemper. He found that the period of incubation varied from 4 to 7 days, and that an increase of internal temperature was the first symptom. An exanthema seldom appears, and only in severe cases. In very mild instances, recovery takes place as early as 6 or 8 days. One attack generally confers immunity. The contagium is contained in

the respective discharges from the nose and eyes, and in the blood. Although it is not destroyed by drying, or by freezing up to  $-20^{\circ}$  C.; its action is weakened by being kept for months in a dry condition. As distemper by inoculation has a mortality of only 10 to 15 per cent., Krajewski recommends inoculation as a prophylactic measure. Laosson made, on dogs and cats, 98 inoculation experiments, which positively demonstrated the contagious nature of distemper. He proved that the disease was identical in both of these animals, and that it can be reciprocally transmitted. He further showed that young cats and dogs became infected almost without exception; that mature and old cats and dogs are less susceptible; that the nasal discharge loses its virulence in 14 days; that the contents of the pustules are ineffective; and that the period of incubation varies from 4 to 7 days. Konhäuser, whose inoculation experiments had a negative result, is of opinion that the contagium is also present in the milk of bitches suffering from distemper.

**Etiology.**—Distemper is a contagious, infective disease, the virus of which has not yet been positively demonstrated. We have not yet been able to obtain pure cultures and their successful transmission. We only know that the contagium is fixed as well as volatile. It is evidently much easier transmitted by cohabitation than by inoculation, and is probably, as a rule, taken from the air during inspiration. Dogs become most frequently affected during the first year of life. Among the 1,378 canine patients which were treated for distemper during one year at the Berlin Veterinary College, 927 (two-thirds) were under 12 months; 269 (one-fifth) were under 2 years; and 182 (one-seventh) over 3 years old. In rare cases, very old dogs may become infected. Puppies only a few weeks old may also suffer from this disease. Contrary to the statements of Krajewski, we have seen an outbreak of distemper in a whole litter of puppies which were between 2 and 3 weeks old. Although one attack generally confers immunity for a considerable time, some dogs become infected several times. We have, for instance, treated the same dog for distemper 4 times in a year.

The chief *predisposing cause* of distemper is chill, brought on, for instance, by exposure in the open air, particularly at night and during cold and wet weather; injudicious washing and bathing; etc. Chill facilitates the penetration of the

virus by weakening the vital forces, or by producing a simple catarrh of the respiratory mucous membranes. A cold of itself can never produce distemper. High-bred, pampered, weak, recently imported, and unacclimatised dogs are specially predisposed to this disease, the occurrence of which is favoured by an unnatural system of feeding and rearing. The old prejudice against meat for the food of young dogs is often the indirect cause of distemper. We may point out that bread weakens the health of dogs, and that meat is the only appropriate food for carnivora. According to the experiments of Bischoff and Voit, dogs which are exclusively fed on bread get into a wretched plight; and cats even die. Progeny predisposed to distemper is likely to be obtained from weakly bitches, and from those which have to bring up an excessively large number of puppies. Considerable hæmorrhage, for instance, by cropping the ears and tail, and rickets, also have a predisposing influence. The supposition that certain breeds become more frequently affected with distemper than others, is probably erroneous; the more likely cause of this apparent susceptibility being the fact that such breeds are more numerous than others. Thus we find in Berlin that more than one-third of all dogs suffering from distemper are pugs; apparently because this breed is at present fashionable, and not because it possesses any special predisposition to distemper.

**Bacteriology.**—Semmer and Laosson found in the blood of affected dogs, a few hours after death, exceedingly slender, small bacilli, which they consider to be the true microbes of distemper. Rabe found in the purulent contents of the pustules, in the nasal discharge, and in the conjunctival secretions of dogs suffering from distemper, bacteria which he regards as the infective agent of distemper. These micro-organisms consisted of globules, the size of which was uniform; but they were so minute that they could hardly be measured. They sometimes lie together in small heaps, or are connected with one another in twos and fours (like *sarcinæ*), or lie in rows of four or five like a string of pearls. They can be stained a dark-blue with methylene-violet. During convalescence, they disappear completely from the nasal discharge, and vary in number proportionately to the severity of the attack. Friedberger confirmed Rabe's statement, but leaves the question of the specific nature of these micro-organisms unanswered for the present. Also, Krajewski noticed the presence of micrococci. Mathis found in the fluids of the body, tissues, sputum, and pustules of dogs suffering from distemper, a specific diplococcus, which he cultivated in neutral or slightly alkaline broth, obtained pure cultures up to the seventh generation, and made successful inoculations with them. The symptoms which appeared after the inoculations, agreed in many respects with those of distemper. For

instance, there was generally a rapid rise of temperature, and pustules appeared on the site of the inoculation or over the whole body. Very young animals frequently died in consequence of this inoculation, which conferred immunity on the survivors. Jacquot and Legrain found in the pus of the pustules numerous motile micrococci, which were from 0.6 to 0.8  $\mu$  in diameter, and which, by uniting, formed diplococci. Inoculations with their cultivations produced local pustules, but no distemper. Marcone and Meloni obtained the same result with the micrococci which they found, and which possessed great similarity to *staphylococcus pyogenes aureus*. Galli-Valerio found in the lungs, brain, spinal medulla and pustules of dogs suffering from distemper, ovoid bacteria, which were 1.25 to 2.5  $\mu$  long, and 0.3  $\mu$  broad, and showed characteristic cultivations. Their inoculations produced in dogs symptoms of distemper, especially those of a nervous type.

Schantyr tried to prove, in a bacteriological work published in 1892, that canine distemper should be divided into three different diseases, which are respectively produced by three morphologically, biologically and pathogenically different micro-organisms. He, however, states that these three forms of distemper are clinically and anatomically so much alike, that they can be distinguished from each other only by an exact bacteriological examination. He named these three forms: "abdominal typhus," "true canine distemper," and "typhoid." We do not feel inclined to give up the clinical unity of canine distemper for such bacteriological problems.

**Occurrence.**—Cats, wolves, foxes, jackals, hyænas, and monkeys become affected with distemper as well as dogs, which suffer from it sporadically, enzootically, and even epizootically. It is almost always present in large towns, in which it is the most common canine disease, and is particularly frequent during certain years. Among the 70,000 dogs which were suffering from internal and external diseases, and which were taken to the Berlin clinic during the years 1886 to 1894, the number of cases of distemper was over 11,000 (one-sixth of all cases). The proportion was about the same in Munich; for among the 2,300 dogs which were received in the Munich clinic during 14 years and which were suffering from internal diseases, 650 were suffering from distemper. This disease can also spread with great rapidity in the country. Jonsson states that distemper raged so violently in Iceland, that there were no dogs seen in large districts of that country. According to our experience, the majority of outbreaks of distemper occur during the summer months. Warmth seems to greatly favour the development and transmission of the contagium.

**Symptoms.**—The symptoms of distemper vary greatly, and are chiefly those of an infectious catarrh of the mucous

membranes of the eyes, and respiratory and digestive organs. These catarrhal symptoms may become complicated with symptoms of severe disturbance of the brain and spinal cord, characteristic exanthema of the skin, and frequently with catarrhal pneumonia. The diversity in the course of the disease has led to the division of distemper into various forms, according to the localisation of the disease, such as catarrhal, nervous, exanthematic distemper; or distemper of the eyes, gastric distemper, pulmonary distemper, etc. These forms often occur in a pure form, that is to say, without any other complication. Thus, the symptoms of an attack of distemper may be essentially those of an infection of the brain, intestines, or eyes, or of an eruption of the skin. As a rule, however, several organs are simultaneously affected. Owing to the want of uniformity in the course of the disease and number of its complications, we can describe only the most important affections of the individual organs.

1. *Initial symptoms.* Distemper is usually first manifested by constitutional disturbance, such as: decreased vivacity, dainty and small appetite, bad temper, trembling and shivering, bristly condition of the coat, warm and dry state of the nose, and tendency to become easily fatigued. In inoculation experiments, there is a considerable increase in the internal temperature, which, even during the period of incubation, rises to  $40^{\circ}$  C.; and, at the commencement of the attack, up to  $41^{\circ}$  C. in slight cases, and to  $42^{\circ}$  C. in severe ones. Owing to the fact that dogs are put under treatment, as a rule, only when the disease has become well-developed, few exact observations have been made on the state of the temperature during the initial stage. It is, however, not improbable that distemper, like other acute infective diseases, commences with high temperature, which lasts only for a short time.

2. *Eye symptoms.* In the large majority of cases, conjunctivitis is the primary ocular symptom. Tears flow from the eyes, and photophobia is present. The mucous membrane of the eyelids becomes highly congested and swollen, and the eyelids turgid. The discharge from the eyes, which is at first serous, soon becomes mucous and purulent. The exuded matter consists of pasty mucus, or dirty yellow pus, which looks like cream or butter. This exudate collects under the lower eyelids, chiefly at the inner canthus of the eye, and soils the edges of the eyelids, upon which it frequently dries into crusts and causes the eyelids to adhere together, particularly



during night. Lesions and ulcers form on the cornea as consequences of the action of the accumulated and decomposing pus ; by the patient wiping and rubbing its eyes with its paws ; and by defective nutrition. The epithelium of the cornea sometimes suffers, more or less, from shallow, flat lesions, which give the surface of the cornea a rough and uneven appearance ; or from smaller but deeper ulcers, especially towards the centre of the cornea. These ulcers, which are often not larger than the head of a pin, are funnel-shaped and penetrate downwards in a straight direction ; their base frequently becomes coated with a pus-like material ; and they heal by proliferation of vessels from the edge of the cornea. In many cases, during their further course, Descemet's membrane protudes, the cornea becomes perforated, prolapse of the iris ensues, and staphyloma is set up ; the usual result being that cicatrisation takes place with the formation of permanent white spots (leucoma) and black pigmentation of the cornea, but rarely with suppurating panophthalmia. Vesicles seldom precede the ulcers on the cornea.

In other cases the cornea, generally of both eyes, becomes affected with a diffuse, parenchymatous keratitis, which renders it, to a considerable extent, opaque, and gives it the appearance of ground glass. These extensive opacities sometimes become developed in a comparatively short time. This affection of the cornea (so-called "distemper of the eyes") is frequently the only symptom of distemper which is present, with the exception of the high temperature. The surface of the implicated cornea is smooth and shining, and the conjunctiva is quite intact. Internal inflammation of the eye, as for instance, an exudative iritis with a fibrinous or purulent exudation into the anterior chamber of the eye, is rarely seen.

3. *Digestive symptoms*, the chief of which are loss of appetite, vomiting, well-marked congestion and dryness of the oral mucous membrane, abnormally great thirst, constipation at first, and diarrhoea later on, in which case the fæces, as a rule, are very foetid, often slimy and frothy, and even bloody (hæmorrhagic intestinal catarrh). Jaundice rarely occurs. The urine frequently contains albumen—especially when the patient is weak, or is in an advanced stage of the disease—and biliary pigments ; but rarely hæmoglobin. In many instances. these are the only symptoms present.

4. *Respiratory symptoms*. The first symptoms are those of

nasal catarrh, such as a nasal discharge which is serous at first, and mucous or purulent later on ; followed by sneezing, panting and nasal pruritus, which causes the animal to wipe its nose with its paw and to rub it on its fore-legs. The pus discharged from both nostrils is often very copious, is sometimes mixed with streaks of blood, and varies in colour from dirty yellow to dirty green. Later on, it may become foetid and even watery. Ulcers are often seen on the nasal mucous membrane. The copiousness of the secretion points to participation of the accessory nasal cavities. The nose often becomes very dry and chapped.

Laryngeal catarrh generally accompanies nasal catarrh, and manifests itself by a cough, which comes on in paroxysms, is at first hoarse and dry, and later on moist and accompanied by a discharge of phlegm. This cough excites the animal to vomit. The catarrh spreads from the larynx to the trachea and bronchi. The resulting bronchitis is followed by increased rate of breathing and manifests itself by cough ; hoarse, sharp, vesicular respiratory murmurs ; and rhonchi. The mucous membrane of the smaller bronchi is frequently catarrhally affected (bronchiolitis or capillary bronchitis), under symptoms of increased respiratory frequency ; difficulty in breathing ; feeble, harassing cough, which may also be set up by percussion of the thoracic walls, by the animals getting up, and by their being taken out of their kennels, and which the patients try to suppress ; humming and whistling sounds ; and dry or moist, crackling or fine râles. Young and weak animals which cannot remove the accumulated phlegm from the bronchi by coughing, become affected with catarrhal pneumonia on account of this excretion becoming drawn into the alveoli. This pneumonia can be recognised by the great increase in the internal temperature ; excessive dyspnœa (pumping up the air and puffing out the cheeks) ; greater or less disappearance of the vesicular respiratory murmur on circumscribed parts of the thorax ; irregular and weak dulness of the percussion-sounds, which may, however, be absent in the case of the inflammatory focus occupying a median position ; occasional tympanitic percussion-sound ; and by the bronchial breathing, which is sometimes audible. The cough is in this case very dull and weak, and the nasal discharge is often foetid. With approaching paralysis of the heart, we may have symptoms of œdema of the lungs, namely : excessive dyspnœa, crackling râles, bubbling rhonchi, tym-

panitic percussion-sound, and gradual decrease in the frequency of respiration.

5. *Nervous symptoms.* Distemper often begins, especially in anæmic animals, with great depression and dulness. Strong animals, however, more usually exhibit symptoms of acute hyperæmia of the brain, such as excitement, high temperature of the head, restlessness, yelping, and even attacks of fury, which give way, later on, to manifestations of cerebral pressure (stupor and a stretched-out condition of the head and neck). These cerebral disturbances may be the only symptoms present in the nervous form of distemper, which at certain times is pretty widely spread. Also, tonic and clonic spasms frequently appear, either generally or locally, and are often confined to particular limbs, which swing backwards and forwards as if affected by chorea. These movements may last even for days, or the patient may have spasms of the muscles of the head, particularly of the facial muscles which receive their nervous supply from the facialis, and muscles of mastication which are supplied by the third motor branch of the trigeminus, in which case, spasms also appear on the lips, cheeks, eyelids, temporal muscles and masseters. Spasm of the masseters often produces a persistent and apparently automatic opening and closing of the mouth. Convulsive contractions of the panniculus, especially in the region of the back, may be seen; and also extremely severe epileptiform convulsions, which are sometimes restricted to the head and neck, but at other times are spread over the whole body. The sick dog, first of all, becomes restless and excited, stares stolidly at objects around him, shakes his head, and runs aimlessly about. Subsequently he is attacked either by spasms of the muscles of mastication, in which case he froths at the mouth, the head and neck are drawn backwards and sideways, and the muscles of the face are convulsed; or he falls down as if from epilepsy, barks, yelps, becomes unconscious, and exhibits tonic and clonic spasms in almost all the muscles of the body, in which case, the sphincters of the anus and bladder become relaxed, and consequently fæces and urine are involuntarily passed. There is a gradual return of consciousness in a short time, usually as soon as 30 to 60 seconds, and the dog manages to get up, although he is very weak. Such an epileptiform attack may pass directly into long-continued coma. Circular and rotatory movements are somewhat rare.

Paralysis also appears, usually as a sequel to the convulsions;

although it may come on simultaneously with them. It seldom occurs at the beginning of the disease. It may be confined to certain groups of muscles, as for instance those of particular limbs; although it very often affects the whole of the hind-quarters, or even the entire body in the form of paresis, especially of the motor nerves, combined with excessive muscular weakness. The sick dog staggers and his hind-quarters sway from side to side, or he becomes incapable of supporting himself on his hind-legs. Frequently, he knuckles over on all four legs, and in severe cases is unable to stand. Permanent paresis of the hind-quarters with paralysis of the bladder and rectum is a frequent sequela of distemper. In some cases, we have noticed paralysis of the muscles of the tongue, which consequently hung loosely out of the mouth, and prehension of food was greatly hindered. During the course of distemper, the following complications have been observed: deafness; amaurosis; cataract in very rare cases; hemiplegia laryngis (roaring); hydrorachis; loss of voice (aphasia), smell and memory; and complete idiocy (hydrocephalus internus). According to our experience, the most common complications are: amaurosis with optic atrophy and chronic hydrocephalus. In a few cases, we have seen unexpected recovery from such amaurosis.

6. *Cutaneous symptoms.* A characteristic pustular exanthema is frequently observed on the inner surface of the thighs and abdomen, and may be the only symptom of distemper in abortive cases, which are the mildest kind of this disease. It first appears in the form of minute red spots, which after twenty-four hours develop into miliary nodules that are surrounded by a red ring. These nodules change into vesicles and pustules, which vary in size from a lentil up to a pea or even a bean, and dry up either into a yellow-brown crust, or burst, and leave weeping, sore spots. Healing takes place with desquamation of the epidermis after about 8 days; leaving bright pigmented, pale-reddish spots, which persist for some time. Generally, there are only a few pustules present. The exanthema may also spread, in the form of a scabby eczema, over the whole body, to the membrane of the external auditory meatus, and less frequently to the mucous membranes of the mouth and eyes. In such cases, the exhalations from the body have a bad smell, and some of the hair falls out. This eruption, contrary to that of sarcoptic mange, is accompanied by only slight pruritus. Intense catarrh of the prepuce may appear

simultaneously with the exanthema. An eruption of wheals (urticaria), spreading over the whole body, has been observed in a few cases.

7. *General symptoms.* The internal temperature, which is usually very atypic, is high during the initial stage, and when local manifestations appear in the lungs, intestines, etc. It often falls below normal with remarkable rapidity towards the approach of death and may be as low as  $36^{\circ}$ ,  $34^{\circ}$ , or even  $32^{\circ}$  C. If the disease runs a protracted course, the patient becomes emaciated; the hind-quarters waste away; the ribs show through the skin; the hair stands on end, and loses its lustre; the bodily exhalations have a very fœtid smell; the eyes are deeply sunken; the mucous membranes are pale; and the patient is weak, staggers when walking, or maintains a recumbent position while in a state of coma.

**Course and Prognosis.**—Distemper has more varieties than almost any other disease. Sometimes it is limited to a pustular exanthema, a parenchymatous keratitis, a mild nervous affection, or a slight catarrh of the mucous membranes of the head or digestive organs. In such, so to speak, abortive cases, a recovery may take place as early as 8 or 10 days; although the disease usually lasts 3 or 4 weeks. With severe complications, especially with those of the nervous system, distemper assumes a protracted course, and is frequently followed by sequelæ, such as paralysis and convulsions, which may persist for months, and even during the whole life. In the lungs, chronic, destructive processes which resemble phthisis and which predispose to tuberculosis, are associated with catarrhal pneumonia. The average mortality is from 50 to 60 per cent. Among unfavourable factors we may mention extreme youth, high-breeding, weakness, anæmia, extension of the process to several organs, convulsions, paralysis, pneumonic attacks, profuse diarrhœa, continued high temperature, emaciation, exhaustion, a depraved condition of the excretory organs, perspiration, and abnormally low temperature of the body. Death takes place from paralysis of the brain (which may have a fatal termination even in a few days), œdema of the lungs, septicæmia, pyæmia, or general exhaustion. Mature age, common breed, and restriction in the spread of the morbid process are favourable conditions. Our experience does not agree with the opinion expressed by some, that the fact of certain forms of distemper being complicated with the

above-described exanthema of the skin is prognostically unfavourable.

**Differential Diagnosis.**—In making our diagnosis, we have frequently to decide the question whether we have to deal with distemper, or with a simple primary catarrh, such as that of the eyes, nose, lungs, stomach, or intestines. To this, a precise answer cannot always be given; because distemper frequently appears as an affection of merely one organ. In general, however, we obtain data sufficient for our purpose from the epizootic character of the disease, youth of the patient, high fever, simultaneous implication of several organs, and unfavourable course of the malady. The demonstration of the pustules of distemper is of great diagnostic importance. The symptoms of cerebral irritation which occur at the beginning of the disease may give rise, especially in the minds of laymen, to the suspicion of rabies. The characteristic aggressive behaviour of animals suffering from rabies is, however, absent from distemper, the further course of which soon settles the matter. The eruption of distemper may resemble that of mange by its becoming greatly extended. The mild character of the pruritus, the presence of pustules on the hypogastrium and inner surface of the thighs, the rapid spreading of the exanthema over the whole body, and the development of other symptoms of distemper, render the differentiation easy. The exanthema of distemper and that of mange, however, often occur simultaneously in the same animal, in which case certain precautions are necessary for making a correct diagnosis. Epileptiform attacks of distemper are essentially distinguished from true epilepsy by their acute character.

**Anatomy.**—The anatomical changes in the *respiratory system* are those of rhinitis, laryngitis, bronchitis, and catarrhal pneumonia. The nasal mucous membrane is either very pale, or greatly congested, swollen, and covered with a thick, purulent greyish-green or dull-red inflammatory exudate which is mixed with coagulated blood, and which collects chiefly between the lamellæ of the turbinate bones and in the frontal sinus. Hæmorrhagic ulcers are sometimes present. The mucous membrane of the larynx and bronchi is also highly congested, swollen, infiltrated with hæmorrhages, covered with pus, and sometimes shows catarrhal ulcers. The large bronchial tubes often

fail to exhibit changes which might have been expected during life; for the hyperæmia and the swelling of the mucous membrane subside after death. The smaller bronchi are, on the other hand, frequently filled up with dirty-grey and even bloody viscid pus. There are spots of congestion on the surface of the lungs, some parts of which may contain but little air, may be entirely free from air, or may contain more air than usual. The catarrhal inflammatory foci of the lungs are characterised by their hardness, absence of air, prominence, and dull-red or copper colour. Their section has a smooth or, in rare cases, a slightly granulated surface, which, when pressed, gives off an opaque, chocolate-coloured fluid that later on becomes dull-red or pale green. In very young animals we may observe the infiltration, into the parenchyma of the lungs, of a fibrinous exudate, which is very soft, and which readily liquefies. This croupy hepatisation frequently has a lobar extension. The inflamed parts of the lungs are sometimes densely studded with numerous suppurating foci, which vary in size from a poppy-seed to more than that of a hemp-seed, or are diffusely infiltrated with pus. Those portions of the pleuræ which cover the affected parts often undergo inflammatory changes. The bronchial glands are swollen and infiltrated with serous fluid, or with pus.

In the *digestive system*, we find the mucous membrane of the stomach and intestines, especially that of the small intestine, congested and swollen, covered with tough, glassy mucus and often infiltrated with hæmorrhages. In other cases, it is very pale, swollen, easily torn, and has a creamy covering. Frequently, the contents of the intestine are sanious; and the mesenteric glands œdematously swollen.

In the *brain*, we often find signs of œdema, such as: anæmia; a relaxed and soft condition of the brain substance; a watery lustre on the surface of sections; flattening down of the convolutions of the brain; and serous effusions into the lateral ventricles and subarachnoideal spaces. In a few cases, we meet with signs of purely venous cerebral hyperæmia, as for instance, great congestion of all the sinuses, venous plexuses and vessels of the pia, and the appearance, on the section surfaces of the brain, of numerous red blood points, which can easily be wiped off. Kolesnikoff found microscopically the brain substance, especially the walls of the vessels, infiltrated with leucocytes; and Krajewski noticed dilatation of the vessels, cellular infiltration of their walls, filling of the perivascular

spaces with lymphoid cells, and immigration of lymphoid cells into the stroma of the brain and into the protoplasm of the ganglionic cells. The changes in the spinal cord, which are not well-marked, consist chiefly in anæmia and slight œdema, especially in the lumbar region. Mazulewitsch, whose observations have been confirmed by Dexler, states that, in acute paralysis, we can find changes of the walls of vessels with an albuminous exudate along the vessels and in the interstitial tissue of the grey matter of the spinal cord; and in chronic distemper, a chronic, localised interstitial myelitis with partial atrophy of the cord. Hadden found groups of emigrated blood-corpuscles in it. According to Trasbot, the spinal cord and its membranes in severe cases are often considerably injected with a sero-fibrinous exudate in and under the arachnoid, and even into the substance of the spinal cord. Hydro-rachis may become developed in chronic cases.

Among other changes, we may mention: decrease in the total quantity of the blood of the body, combined with a certain degree of hydræmia; opacity and fatty degenerations of the epithelium of the liver and kidneys; dull yellow discoloration of the muscular tissue of the heart in consequence of dust-coloured cloudy swelling and fatty degeneration of its fibres; œdematous swelling of the lymph glands; tarry and greasy condition of the blood (in complications with septicæmia); and capped elbow and capped hock from decubitus.

**Therapeutics.**—The treatment of distemper is essentially symptomatic. There are, however, medicines which are able to subdue, *in loco affectionis*, the contagium of distemper, after it has entered the body. Of such medicines, calomel is a good instance. According to our experiments, this drug can be used with advantage in every case in which the digestive organs have been the starting point of the infection. We obtained, especially in the initial stage of the so-called gastric form of distemper, good results from its administration two or three times a day, in doses of 0.05 gramme. Calomel acts as a disinfectant of the mucous membrane of the stomach and intestines. The same may be said of the disinfection of the respiratory mucous membrane by means of inhalations of creolin ( $\frac{1}{2}$  to 1 per cent.), which have an equally good effect in the bronchial form of distemper. Our success in the treatment of the bronchial and pulmonary forms of distemper has been greatly enhanced by the adoption of creolin as a local disinfectant of the respir-



atory mucous membrane; although in this disease we are often compelled to use a purely symptomatic treatment, which varies according to the localisation of the malady.

1. When the *eyes* are affected, their treatment has to be carried out according to the rules of ophthalmology. A solution of  $\frac{1}{2}$  to 1 per cent. of creolin is an admirable application for suppurating conjunctivitis and ulcers of the cornea. In the former affection, good results may be obtained with a  $\frac{1}{2}$  to 1 per cent. solution of sulphate of zinc, or in obstinate cases, with a  $\frac{1}{2}$  to 2 per cent. solution of nitrate of silver; and in the latter, with a 2 per cent. solution of boric acid or quinine. We may treat parenchymatous keratitis with  $\frac{1}{2}$  per cent. solution of sulphate of atropine. A 1 to 2 per cent. solution of hydrochlorate of cocaine (5 minims to be dropped into the eyes every 3 hours) may be employed to combat the abnormal sensitiveness of the mucous membrane of the eyelids, which causes the patient to wipe and rub his eyes, and which, thus, often induces the formation of purely traumatic ulcers. In cases of blenorrhœa, we may cauterise the inflamed mucous membrane with a point of pure or diluted nitrate of silver, with one of bluestone, or in very obstinate cases, with the thermocautery after a previous application of cocaine. Opacities of the cornea of some standing may be more or less removed by rubbing them with red precipitate ointment (1 to 20 cir.). Staphyloma and entropium require surgical treatment.

2. The affections of the *digestive apparatus* may be treated in different ways. An emetic ( $\frac{1}{100}$ th to  $\frac{1}{200}$ th of a gramme of apomorphine hydrochlorate in from 1 to 5 grammes of water, subcutaneously) sometimes renders good service at the commencement of the disease. As a stomachic, we can recommend hydrochloric acid in doses of a drop by itself in water, or combined as follows: hydrochloric acid 1 part, tincture of gentian 4 parts, water 60 parts; half or a whole teaspoonful to be given 3 times a day. We may also administer *tinctura rhei aquosa* and *vinosa* (in doses of a teaspoonful); decoctions of cinchona bark (1 to 15, a tablespoonful at a time, by itself or combined with hydrochloric acid); bismuthi subnitras (0.1 to 0.2 gramme); extract of meat (2 to 5 grammes); wine, quinine wine, condurango wine (from a teaspoonful to a tablespoonful), etc. Opium with small bits of ice is indicated in persistent vomiting. Diarrhœa may be treated with tinct. opii, 0.5 to 3 grammes; powdered opium, 0.1 to 0.3 gramme; tannin, 0.2 to 0.5

gramme; and in very obstinate cases with nitrate of silver, 0.02 to 0.05 gramme in pills. In dysenteric diarrhœa we give opium with mucous remedies, as for instance, tinct. opii 14 parts, acaciæ gummi 15 parts, aqu. fœniculi 300 parts, 3 times daily a teaspoonful or a tablespoonful.

3. In affections of the *respiratory organs*, we should first of all employ the remedies which we have mentioned for the relief of laryngitis, bronchitis, and catarrhal pneumonia. For cough, we get good results with morphia and codeine, which we may prescribe as follows: R. morphinæ hydrochloras 0.1 gramme, aqu. amygdal. amar. 10 grammes, aqu. destill. 150 grammes, M. a teaspoonful or a tablespoonful 3 times daily. Or: codeinæ phosphor. 0.3 gramme, syrupi althææ 10 grammes, aqu. fœniculi 150 grammes, M. a teaspoonful or a tablespoonful every 3 hours. Besides this we recommend moist, warm fomentations over the larynx and thorax. The affection of the bronchi requires the employment of expectorants, such as apomorphinæ hydrochloras (0.01 gramme *pro die per os*); tartar emetic (only for strong animals) and sulphurated antimony (0.02 to 0.05 gramme); chloride of ammonium (0.1 to 0.5 gramme); vinum antimoniale (in doses of a drop); infusum senegæ (a teaspoonful up to a tablespoonful); and ipecacuanha (0.02 to 0.05 gramme), etc.

4. For spasms caused by disturbance of the *central nervous system*, we prescribe potassium bromide and sodium bromide (1 to 25 of water, a teaspoonful or tablespoonful, 3 times daily); sulphonal (1 to 4 grammes); and chloral hydrate (0.05 to 5 grammes) in mucilage; or subcutaneous injection of morphia (0.02 to 0.1 gramme). We treat paralysis and weakness by stimulants, such as coffee (5 to 10 per cent. infusion), coffee with brandy, beef tea, extract of meat, wine, camphor, (spirit. camphor. or linimentum camphoræ 5 to 10 grammes rubbed into the skin); ether (1 gramme every hour, subcutaneously); caffeine ( $\frac{1}{2}$  to 2 grammes subcutaneously); hyoscine (0.005 to 0.02 gramme subcutaneously); atropine (0.01 to 0.05 gramme); veratrine (0.001 to 0.005 gramme); strychnine (0.001 to 0.003 gramme subcutaneously); and electricity (Spamer's machine).

5. The *fever* (rise in the internal temperature) should be specially treated only when it attains a great height, or when by its persistent presence it threatens to become dangerous to the more important organs of the body, especially the heart. The initial fever, and any slight rise of temperature, say up to

40° C., require no antipyresis. As febrifuges we prefer antipyrine (0.5 to 2 grammes) and antifebrine (0.25 to 1 gramme); because they reduce the temperature with great certainty and in a short time ( $\frac{1}{4}$  to 1 gramme every hour until the temperature becomes normal). The effect of quinine is not so certain as that of antipyrine or antifebrine.

6. The *skin* affection requires little or no treatment. Moist sore places may be dried with powders, such as 1 part of oxide of zinc to 10 of talc. The application of glycerine to the crusts of the dried-up pustules will hasten their removal.

As regards diet, we should give easily digested nutritious food (the best kind of which is raw meat minced or scraped), milk, and beef tea. The affected animals should be segregated from the healthy.

**Distemper of Cats.**—Krajewski proved, by the experimental transmission of the distemper of cats to dogs, and *vice versa*, that the two diseases are in every respect identical. Feline distemper may occur enzootically. Its symptoms are precisely the same as those of canine distemper. Nervous attacks, however, appear to be rarer in cats than in dogs, and may be entirely absent. The initial symptoms consist of loss of appetite, retching, vomiting, depression, etc., followed by suppurating conjunctivitis, purulent nasal discharge, sneezing, coughing, snorting, difficult breathing, diarrhoea, screaming, weakness, anæmia, and, towards the end, symptoms of coma with a fall of internal temperature. There seems to be no exanthema. On *post mortem* examination, we find changes similar to those in dogs. Contrary to the statements of other observers, we have found on several occasions, especially in cats, extensive croupy pneumonia. With regard to therapeutics, we would advise exactly the same treatment for cats as for dogs, with the exception of the omission of carbolic acid, which has a very poisonous effect on cats. The doses appropriate to these animals is about the fifth of those usually given to dogs.

## MALIGNANT CATARRHAL FEVER OF CATTLE.

Malignant catarrhal fever of cattle—Pseudo-malignant catarrhal fever of sheep—Malignant catarrhal fever of horses.

**Etiology.**—Malignant catarrhal fever is a disease which occurs only in cattle and buffaloes, and has nothing in common with human diphtheria. We possess no exact knowledge respecting its contagium. Its contagiousness, in comparison with that of other infective diseases, is very slight. Direct infection has been observed only in exceptional cases, and all the inoculation ex-

periments which have been made, have given negative results. Its cause is probably a cowshed miasma, which becomes developed chiefly in damp, dirty sheds with defective drainage and bad ventilation. According to Frank, this disease is generally found on moderately moist, loose soil, and not on very wet or entirely dry subsoil. It is possible that the disease may also spread by means of intermediate bearers, as for instance, sheep (Möbius), or, as we have observed, cattle-dealers.

Malignant catarrhal fever is usually sporadic; although its appearance as an enzootic throughout large cow-sheds and even villages is not rare. In some places it is a permanent enzootic, and may break out during several successive years in the same shed. A great number of such cases are on record. Sump saw an instance of the disease remaining stationary for 25 years on a gentleman's estate, during which time 225 cows were attacked with the result of only 3 recoveries. Although young and well-nourished animals are most susceptible, many of mature age also suffer. It appears that the disease usually occurs in spring. We know nothing certain as to the influence of locality on this disease, which is found in high-lying places as well as in valleys and plains. The same remark applies to the influence of surface water. Bugnion and Frank state that the period of incubation is 3 or 4 weeks. Chill, which was formerly regarded as the original cause, has, as in other diseases, only a predisposing action. An animal may become repeatedly affected within a short period.

It has always been recognised that the disease assumes various forms, according to the respective localisation and intensity of the affection. Thus we speak of a nasal, abdominal, exanthematic, benign, and malignant form. The protean character of this disease in different animals has, for a long time, given rise to inexact and confused ideas respecting its pathology.

**Symptoms.**—The symptoms of malignant catarrhal fever are exceedingly various and changeable. On the whole, this disease is a grave general affection, in which the mucous membranes of the eyes, respiratory organs, cavities of the head, alimentary canal, and urinary and sexual organs are attacked more or less severely, in a catarrhal, croupy or diphtheritic form; and there is severe nervous disturbance, especially of the functions of the brain. Usually, several organs or several functions are simultaneously affected, but occasionally only one suffers.

1. *Initial symptoms.* The disease generally begins rapidly with rigors and high fever, the temperature being between  $40^{\circ}$  and  $42^{\circ}$  C., even on the first or second day. The animal exhibits great torpor, supports its head on the manger, or holds it very low. The upper part of the head, especially the region of the frontal sinus and the base of the horns, is hot to the touch, and continued muscular tremor is noticed over the whole body. The hair is rough and stands on end, and the back is frequently arched. Even after the first few days, the patient sometimes becomes greatly emaciated.

2. *Eye symptoms.* Profuse lachrymation is generally the first well-marked symptom, in which case tears flow abundantly over the cheeks, and there is swelling of the eyelids, excessive congestion of the conjunctiva, and considerable photophobia, with consequent closure of the eyes. The swelling of the eyelids may lead to well-developed ectropium. In its further progress, this conjunctivitis becomes complicated with ordinary diffuse keratitis, in which case, the cornea becomes affected with an opacity which extends from the periphery inwards, and is at first dull and smoky, and later on milk-white. Ulcers on the cornea are rare. We may also have inflammation of the iris (iritis exsudativa et adhæsiva), in consequence of which there is an exudation into the anterior chamber of the eye, and adhesion of the iris to the capsule of the lens. Resolution may take place; or the changes may lead to perforation of the cornea, complicated with prolapse of the iris, and atrophy of the eye-ball, which will be followed by permanent blindness. Persistent opacities of the cornea (leucoma) frequently occur. This affection of the eyes throughout its entire course closely resembles equine periodic ophthalmia.

3. *The respiratory symptoms* consist at first of catarrhal, and later on, of croupy and diphtheritic (sloughing) inflammation of the mucous membrane of the nose, larynx, bronchi, and accessory cavities of the head, with swelling of the lymph glands. In the early stage, the nasal mucous membrane is of a bright red or purple colour, but afterwards it becomes coated with a croupy and diphtheritic material which leaves ulcers behind. This inflammatory affection of the nasal mucous membrane is accompanied by a nasal discharge, which at first is sero-mucous, but soon becomes purulent, lumpy, mixed with streaks of blood, discoloured, and finally foetid. Respiration is difficult, frequently stertorous, rattling, wheezing, and combined with mucous râles. The dyspnoea may turn into regular attacks of

suffocation on account of the lumen of the larynx or nose becoming plugged up with croupy membranes. Croupy-diphtheritic masses are sometimes coughed up. When the inflammation spreads from the frontal sinus to the cavities of the horn-cores, inflammation of the matrix of the horn (which represents the periosteum of the processes of the horns) may ensue. The horns will then be very painful to the touch, and can be easily removed, or may even become shed.

4. *Digestive symptoms.* In severe attacks, the appetite decreases from the commencement, or is entirely suppressed. In slight cases, or when the disease is located in other organs, the appetite often remains good for a considerable time. During the first few days, the oral mucous membrane is considerably congested, dry, and hot. Later on, the animal becomes salivated from stomatitis, frequently to such an extent that the saliva flows in long strings to the ground. In such cases, the mucous membrane is infiltrated with hæmorrhages, and manifests, especially on the palate, cartilaginous pad of the upper jaw, and cheeks, erosions and insulated diphtheritic deposits, which vary in colour from yellow-grey [buff] to yellowish-white, and which leave ulcers behind after they are cast off. The necrotic inflammation may also spread to the skin of the muzzle and nose, in which case, the entire anterior part of the head may become swollen. Under these circumstances, the oral cavity emits a very bad odour. Further on, we observe constipation, alternating with diarrhœa, and symptoms of uneasiness as if from colic. The fæces are foetid, and resemble yeast or liquid manure. They are frequently mixed with blood, and not rarely contain membranous shreds. Frank states that croupy deposits from the intestinal mucous membrane are sometimes passed along with the dung. Tenesmus is present at the same time, and the mucous membrane of the rectum is considerably swollen. The secretion of milk usually stops even during the first few days of the attack.

5. *Urinary and genital symptoms* are those of nephritis and cystitis. There is severe strangury. The urine often contains blood and the characteristic products of inflammation of the kidneys, such as urinary casts, epithelium of the kidneys, leucocytes, and albumen. According to Frank, its reaction is often acid. The vaginal mucous membrane is hyperæmic, swollen, and sometimes shows diphtheritic deposits, ulcers and a mucous discharge. Pregnant animals which are severely affected, frequently abort.

6. *Nervous symptoms* play the chief rôle in many cases of this disease, and consist, as a rule, in those of hyperæmia and inflammation of the brain. The animal becomes greatly excited and very restless, climbs up on the rails, bellows loudly, shoves up against its fellows, and keeps to one side. It assumes a wild, staring look, and frequently gets into a regular frenzy ; or they may exhibit attacks of extremely varied kinds of spasms, such as convulsions, epileptiform fits, rolling of the eyes, and even seizures resembling trismus. The spasms often change later on into paralysis, or merge into a condition of general weakness and increasing coma, in which case the disease ends in death, preceded by a fall in the internal temperature.

7. *The skin* becomes occasionally affected, in which case we find an eruption of vesicles or nodules, falling off of the hair, and, subsequently, severe desquamation of the epidermis, for example, on the udder, inner side of the joints, interdigital space, inner surface of the thighs, throat, and back. The hoofs may become shed in rare instances. Strebel states that a bilateral inflammation of the hock has sometimes been observed.

**Anatomy.**—We find as much variety in the anatomical changes as in the symptoms, according to the duration, localisation, and severity of the disease. Slaughtered animals show chiefly initial changes, such as catarrhal or croupy processes ; and those which have died from the disease, true diphtheritis of the mucous membranes. The following are the most important points that have to be considered in the examination of the respective organs, which are by no means simultaneously affected in all cases.

The mucous membrane of the *nasal cavity* is of a brownish-red or purple colour ; and is swollen, infiltrated with hæmorrhages, covered with whity-yellow, croupy masses, and is ulcerated. The sloughing process sometimes spreads to the turbinate bones and ethmoidal cells. The mucous membrane in the frontal sinus is also swollen and covered with pus ; there is an accumulation of pus in the cavities of the horn-cores ; the fleshy walls of the horns are inflamed ; the mucous membrane of the larynx and bronchi shows simple catarrhal, or hæmorrhagic and croupy inflammation ; the croupy coating often extends into the small bronchi ; and the lungs very frequently exhibit interstitial emphysema, and also œdema in fatal cases.

The mucous membrane of the *oral cavity* is of a purple colour and swollen, particularly near the teeth. It is said that vesicles,

which rapidly change into superficial erosions, may be seen on it. These erosions, which are not rare, may be larger than a sixpence. In severe cases, the mucous membrane, especially of the soft and hard palate, becomes covered with insular whitish-yellow membranes, which frequently cover the greater part of the pharyngeal cavity and may extend even into the œsophagus. The mucous membrane of the pharynx generally shows the same changes as those of the oral cavity. The mucous membrane of the stomach and intestines is congested diffusely and in spots, swollen, and often covered with diphtheritic deposits and ulcers, which may leave scars after healing. Peyer's patches and the solitary follicles are usually swollen. Thus, the aspect of the disease in many cases may closely resemble that of rinderpest.

The *cavities of the skull* are rich in blood, especially in the cerebral meninges; and there are slight hæmorrhages and œdema in the brain and its membranes. Inflammation of the brain has also been observed. We find similar changes in the spinal cord. The kidneys are inflamed and show fatty degeneration. In the pelvis of the kidney we sometimes find croupy deposits which may extend to the ureters and bladder. The mucous membrane of the bladder is inflamed in extremely varied degrees, is often covered with croupy membranes, and infiltrated with hæmorrhages. Among general changes we may observe: hæmorrhages throughout the entire body, especially in the heart, under the serous membranes, in the mesentery and omentum, and under the skin; fatty degeneration and swelling of the liver; fatty degeneration and discoloration of the muscles; rapid putrefaction of the cadaver; dark-coloured and thick blood; accumulation of sero-sanious fluid in the cavities of the body; slight inflammation of the pleuræ; etc.

**Course and Prognosis.**—The course of malignant catarrhal fever is sometimes acute and even peracute; at other times subacute, and even chronic. Death may take place as early as from 3 to 5 days after the attack, although recovery may be delayed for several weeks. The disease lasts on an average 3 or 4 weeks. Relapses are so frequent that an acute seizure may take place as late as the second or third week.

During the first few days, we may often observe an apparent improvement, which may be quickly followed by a severe exacerbation. The period of convalescence is always more or less protracted. In mild cases, complete recovery takes place in



about a month ; but may be retarded for double or treble that period in severe attacks, except when the course of the disease has a rapidly fatal termination.

Prognosis is always very doubtful. The mortality varies from 50 to 90 per cent. Among 76 cases, observed by Bugnion, only 7 recovered. All the others were slaughtered on the third or fourth day after they became ill. Frank saw only 6 per cent. of the treated animals recover. Unfavourable factors are : simultaneous extension of the disease to several organs, or to all the organs of the body ; development of the inflammation into necrosis of the affected mucous membranes ; and grave cerebral, pulmonary and intestinal attacks. Besides the previously described changes in the eyes, we have, as a sequela, the development of chronic catarrh of the sinuses of the head.

**Differential Diagnosis.**—Most important of all is the differentiation of malignant catarrhal fever from rinderpest, which is by no means easy on all occasions. The following are points to be considered : 1. The contagiousness of malignant catarrhal fever is as slight as that of rinderpest is great. 2. In malignant catarrhal fever, the best-marked symptoms are those of the eyes and respiratory organs (stridor and nasal discharge) ; and in rinderpest, those of gastric disturbance. The same remark applies to the *post mortem* appearances of the respective diseases. 3. The course of malignant catarrhal fever is, as a rule, not so acute as that of rinderpest. The differentiation becomes much more difficult when several animals are affected with malignant catarrhal fever in the same shed, and when the intestinal canal is specially affected with simultaneous implication of the vaginal mucous membrane. Here we may try inoculation, the negative result of which will be a certain sign of catarrhal fever. This disease may also be mistaken for meningitis, dysentery, periodic inflammation of the eyes, and benign rhinitis fibrinosa (Berndt), in which we find neither opacity of the cornea nor any nervous symptom. This rhinitis is, however, infectious, and sometimes appears as an epizootic.

**Therapeutics.**—Frank, Hink and others have recently called attention to the importance of thorough disinfection and draining of the subsoil in those cow-sheds in which the disease is stationary. It has also been recommended to render the floor of the shed waterproof. The infective nasal discharge should

be frequently removed and thoroughly disinfected. Segregation is also advised. No remedy has as yet been found. In former times, blood-letting, washing the skin with vinegar, inhalations of vinegar, sawing off the horns, trepanning, and the administration of tartar emetic and camphor were recommended. Recently, many authorities, such as Frank, Hink and Zündel, say that they have obtained successful results with the internal administration of creolin and carbolic acid (5 to 10 grammes), and with external applications of these agents in the form of washes, inhalations, and ointments. Hink strongly recommends washing out the nasal cavity with a 2 per cent. solution of creolin. When severe cerebral symptoms are present, we recommend the continued application of cold to the head, as for instance, by means of bags of ice and cold douches. This treatment has been in vogue for a long time. The other symptoms should be treated symptomatically with antipyretics, alkalies, expectorants, emollients, etc.

**Pseudo-malignant Catarrhal Fever of Sheep** is a contagious infective disease which possesses an extraordinary likeness to distemper of dogs, and which is characterised by an affection of all the mucous membranes, especially those of the nasal cavity, eyes, and respiratory organs. The investigations of Friedberger point to the probability that the malignant catarrhal fever of sheep is caused by a contagium which may remain in a shed for several years, and which can evidently be transmitted to a certain distance; because animals which were in healthy and separate sheds have become infected. Audum believes that he has found the contagium in the form of a bacillus of from 1 to 3  $\mu$  long, and of the thickness of a tubercle bacillus. The disease usually occurs enzootically, and in some instances, epizootically. It is with justice greatly feared. Formerly it was thought that this disease was caused by simple coryza becoming "malignant," "degenerating," or passing into "glanders." Other authors speak of an epizootic form of benign catarrhal fever. This disease is not identical with the catarrhal fever of cattle.

The disease begins with symptoms of suppurating nasal catarrh. Aropy mucous discharge collects round the nostrils, which often become glued together, and from which a muco-purulent and (according to some writers), sometimes, a foetid substance can be squeezed. The nasal mucous membrane is highly congested and swollen, and symptoms of laryngitis (coughing), bronchitis, and in severe cases even symptoms of bronchial pneumonia or pneumonia caused by foreign bodies may appear at the same time. Suppurating blepharitis, purulent conjunctivitis, keratitis, and even ulcers and abscesses of the cornea are also seen. The eyelids are reddened, swollen, and glued together by a greasy, purulent substance. The eyelashes below the inner canthus are wet and soiled with a yellowish-brown greasy discharge. The conjunctiva varies in colour from orange to cinnabar-red, is swollen, and often secretes

enormous masses of purulent secretion (blennorrhœa). The skin near the orifice of the sheath suffers from a suppurating dermatitis, accompanied by catarrh of the prepuce. We find symptoms of intestinal catarrh, such as constipation, diarrhœa, straining to defecate, and suppressed appetite. The general health is greatly upset; and the animal becomes depressed and anæmic, rapidly loses condition, is slightly feverish, and staggers and reels when walking. Finally, the patient, being too weak to stand, falls down and lies on the floor in deep sleep, which sometimes is interrupted by symptoms of approaching death, such as clonic spasms of the muscles of the limbs and trunk, grinding of the teeth, etc. The course of the disease is somewhat acute, and prognosis is very unfavourable, especially in lambs.

On *post mortem* examination we find the mucous membrane of the nasal cavity, particularly its upper parts, to be congested in a more or less diffuse and ramiform manner, especially the mucous membrane of the turbinate bones, which varies in colour from scarlet to very dark red or chocolate, and is swollen, very shiny, and covered with a purulent secretion, which in some parts may look like frog-spawn, and in others may be creamy, lumpy, caseous, and of a dirty dull-yellow colour. Erosions and ulcers are sometimes found after the removal of this coating. In the other air-passages the changes are those of catarrhal bronchitis, inflammatory œdema of the lungs, broncho-pneumonia, and pneumonia caused by foreign bodies. The liver is pulpy, brittle, infiltrated with fat, and of a clay colour. The epithelium of the kidneys shows a high degree of fatty degeneration; the brain is slightly œdematous; the muscles of the heart are relaxed, brittle, and of a dull-red colour; the blood is very thin or manifests no tendency to coagulate; the muscles of the body are pale; the cadaver is greatly emaciated; and ecchymoses are found in various organs. *Prophylaxis* consists in the respective separation of the healthy and of those which are but slightly affected; in thorough disinfection of the sheds; and in the supply of good nourishing food, such as mashes and bruised oats. Tonics, such as sulphate of iron, and bitters in the form of electuaries may be given in mild cases. It is best to slaughter those animals which are severely affected; for they are generally beyond hope of recovery.

**Malignant Catarrhal Fever of Horses (?)**.—During an outbreak of catarrhal fever in cattle, Hable saw two horses which were affected with a disease that very closely resembled it. One of these horses had a purulent discharge from the nose and mouth, opacity of the cornea, swelling of the eyelids, lachrymation, difficulty in swallowing, considerable swelling of the pharyngeal lymph glands, swelling of the head, and inflammation of the lungs. This animal died on the fourth day of the attack. The other horse presented similar symptoms; the opacity of the cornea and swelling of the eyelids being particularly well-marked. He recovered, but remained blind in consequence of opacity of the cornea.

## ENZOOTIC ABORTION.

**Etiology.**—Abortion occurs as an infective disease among cattle, and less frequently among sheep, goats, pigs, and horses, at certain times, and in certain stalls, pastures, and districts. On some estates it appears every year. Although the nature of the contagium is unknown, we may infer that the disease is of an infective character, from the fact that it occurs enzootically and sometimes even epizootically; and that ordinary abortion is observed only sporadically, in consequence of mechanical injuries, acute feverish attacks, chill, poisoning, and partaking of food and drink which contain fungi and other noxious material.

The infection, which seems to proceed from a cow-shed miasma, has been proved to exist in the excretions of the genital organs, and in the liquor amnii of cows which have aborted, and to be capable of being transmitted to healthy pregnant cows by means of such fluids. Consequently, the disease is often introduced into healthy herds through the purchase of cows from infected places. Bräuer was the first to experimentally produce enzootic abortion, by successfully transferring, in 11 cases, the vaginal mucus of cows that had aborted, to the vaginal mucous membrane of healthy cows. In this experiment, abortion took place in from 9 to 21 days after the inoculation. Lehnert in this manner twice produced abortion, which appeared in one case after 12 days; and in the other, after 20 days. Trinchera set up suppurating vaginal catarrh, followed by abortion in from 9 to 13 days, in healthy cows by the inoculation of the purulent vaginal mucus of an affected cow on the vaginal mucous membrane of healthy cows; and obtained similar results with the material scraped from the surface of the chorion of an immature fœtus. Kilborne and Smith observed suppurating vaginal catarrh in cows and mares after inoculations of cultivations of bacilli obtained from the vaginal secretion of mares which had aborted. Turner succeeded in producing abortion in pregnant mares by means of the vaginal injection of cultivations made with the membranes of the ova of mares that had aborted, and with the secretion from the genital organs of foals suffering from umbilical pyæmia, which, according to him, has the same origin as epizootic abortion. Experience has amply proved that epizootic abortion is highly contagious, and that it can be transmitted not only

directly, but also through intermediate bearers, such as liquid manure, litter, servants, veterinary surgeons who had a short time previously removed an after-birth, and even stud bulls. Hence, the disease usually spreads from one cow to its neighbour, and so on. We may also frequently trace the occurrence of an outbreak to the fact of the liquid manure remaining stagnant in the neighbourhood of the implicated cows.

Such factors as defective nourishment, exclusive stall-feeding, continued in-breeding, and preceding bad seasons, which were formerly considered to be the chief causes of epizootic abortion, have really only a predisposing influence. By weakening and relaxing the system, they facilitate the penetration of the virus into the body, and favour its development. This idea is strengthened by the fact that Strebel saw from 20 to 60 per cent. of all the pregnant animals abort in the Canton of Freiburg during the wet years of 1878-1879. The fact that residence in insanitary and dirty stalls is not indispensable to the production of the disease, is proved by the repeatedly observed occurrence of infective abortion in perfectly normal, newly built, and absolutely clean sheds, and even in model sheds, and among the Hungarian steppe-cattle, which live in the open during the whole year (Kocourek). The mere putrefaction of the after-birth cannot of itself originate the disease; because the occurrence of enzootic abortion is extremely rare in comparison to that of the retention and putrefaction of the after-birth in cattle.

We know nothing positive respecting the pathogenesis of infective abortion. It is probable that the contagium penetrates through the vaginal canal and the os uteri into the uterus in a manner similar to that adopted by the septic virus of puerperal fever, with consequent implication of the membranes of the ovum, thereby producing abortion. At least the fact that in these cases of abortion, the foetus usually is dead, seems to confirm this theory. The death of the foetus is a further incentive to abortion. We are not yet able to decide whether or not the contagium can enter the blood by means of the air passages or intestinal canal. Galtier, Poncius and Ory consider that this form of abortion is due to a general infection of the mother, and that she transmits the disease to the foetus. These authorities succeeded in artificially producing this general disease by inoculation of morbid matter obtained from the aborted foetus, and from the membranes of the ovum, with subsequent abortions in cows, pigs, sheep and goats. Biot states

that the cause of the abortion is an infective inflammation of the serous covering of the uterus. Nocard asserts that the contagium, which, he says, consists of different kinds of bacteria, is to be found between the mucous membrane of the uterus and the foetal membranes, and that it remains permanently in the uterus, without having any injurious influence on the maternal mucous membrane; and that it attacks the foetal membranes after conception has taken place, and thus invariably produces abortion. This contagium is also supposed to cause sterility by rendering acid the fluid of the uterus, which, when it is in this condition, kills the spermatozoa that penetrate into this organ.

Several observations made by Danish veterinary surgeons tend to prove that animals which have passed through the disease become immune; for this kind of abortion remains in cow-sheds, as a rule, only for from 2 to 4 years, if no fresh infected cows are brought into the sheds (Sand).

**Symptoms.**—This form of abortion occurs in cows usually during the third or seventh month of pregnancy; in mares, during the fourth or ninth month; and in pigs during the tenth or twelfth week. The premonitory symptoms are: swelling of the udder; great congestion of the vaginal mucous membrane; dirty-red (whity in mares), catarrhal, muco-purulent discharge from the vagina; occasional formation of pimples the size of a millet seed on the mucous membrane; decreased yield of milk; and a colostrum-like condition of the milk. Abortion generally occurs about 3 days after the appearance of the discharge, with strikingly mild general symptoms. The foetus, as a rule, is born dead. A persistent vaginal discharge often remains for a considerable time after the abortion, in consequence of which the cow may suffer from continued bad health, and may become sterile.

**Therapeutics.**—Prophylaxis is the chief thing to be attended to in cases of enzootic abortion. If the act of abortion once begins, it cannot be stopped by the administration of opium or any other remedy. The most important and frequently the only effective measure is to transfer the pregnant cows to another shed as soon as possible; or, if practicable, to turn them out to grass and to rigidly isolate them as regards feeding and tending. It is also imperative to completely remove and to render innocuous every after-birth and dead foetus. Each stall and the

entire stable should be thoroughly disinfected ; \* the patients should be disinfected by washing the genital organs † with a watery solution of creolin or carbolic acid, and injecting a similar solution ( $\frac{1}{2}$  to 2 per cent.) into the vagina and uterus. The external genital organs and the vagina of pregnant cows should be disinfected in the same way. Grazing sows which might infect themselves by the vaginal discharge of affected animals, and sows which have aborted, should be kept away from the pastures for a few weeks (Gassner). In order to decrease the predisposition to the disease, we might try to strengthen the constitution of the animals by exercise and nitrogenous food.

Abortion often reappears in spite of the thorough disinfection of sheds and animals, and in spite of good feeding and attendance. Consequently, Bräuer attempted to protect the animals against the disease by means of subcutaneous carbolic injections (two or three Pravaz syringefuls of a 2 per cent. solution) under the skin of the flanks, every fortnight, from the fifth to the seventh month of pregnancy. He states that he obtained good results with these injections in a series of cases. Some of the recent reports on the efficiency of this method are positive ; others negative (Schleg). From a purely theoretical point of view, it seems improbable that a good effect could be obtained with carbolic acid ; because that agent quickly changes in the body into sulpho-phenic acid, which is inert. It is probable that the alleged favourable results were due to the simultaneous disinfection of the external genital organs.

The affected animals should not again be used for breeding purposes until the vaginal discharge has completely stopped. A change of the bull sometimes appears to exercise a favourable influence on the epizootic ; probably because it freshens up the breed and improves the constitution of the cows, and also, perhaps, by excluding a former intermediate bearer. As freshly-bought breeding animals may be intermediate bearers, they ought at first to be kept separate from the rest.

[The following notes have been compiled from the article on "The Etiology of Epizootic Abortion" (*Journal of Comparative Pathology and Therapeutics*, June, 1897), by Professor Bang of Copenhagen, who has thrown much new light on this subject :—

\* Bang advises the use of quicklime ; as, besides being efficient, it neither taints the milk, nor diminishes the value of the manure.—Tr.

† Disinfection of the glans penis and sheath of the suspected male should not be neglected.—Tr.

**Definition.**—Enzootic (or epizootic) abortion is a symptom of a specific infective catarrh of the uterus, which catarrh appears to be a purely local affection; because, in its natural course, it is accompanied neither by fever, nor by constitutional disturbance, and gives rise to no morbid changes in the tissues, with the exception of the uterus. Abortion is not an invariable symptom of this infective catarrh.

**Etiology and Bacteriology.**—Bang and Stribolt have proved by the inoculation of pure cultures that the disease is set up by a very small non-motile bacillus, which contains one, two, or more rarely three, roundish or elongated granules. The bacilli vary greatly in length; the maximum being about that of a tubercle bacillus. They readily take Löffler's methylene blue stain. They are most abundantly found in the yellow exudate which is met with in this disease between the mucous membrane of the uterus and the foetal membranes. In cultivation, the bacillus presents the peculiarity of thriving best either when the percentage of oxygen is somewhat less than it is in the air (21), or when it approaches but does not reach 100. In atmospheres of intermediate percentages of oxygen, it thrives more or less badly, or ceases to grow. The examination of mummified fetuses has shown that the bacilli can remain virulent for at least 7 months.

**Anatomy and Progress.**—The only characteristic *post mortem* changes are: (1) an odourless, dirty-yellow, flocculent, slimy and more or less thin exudate which is to be found between the uterus and foetal membranes; and (2) oedema of the sub-chorial connective tissue, which is consequently abnormally thick, slimy, gelatinous, and brittle. The progress of abortion does not appear to be dependent on morbid changes in the foetus, in which the special bacillus may or may not be present. The catarrh of the uterus may exist without causing premature delivery. The bacilli may become transmitted to the foetus and may thereby give rise to its death *in utero*, with mummification in some cases.

**The Period of Incubation** (*i.e.*, time taken by the bacilli to set up inflammation in the uterine mucous membrane) may extend to 10 weeks, or, perhaps, even longer.

**Transmission.**—The theory that the bull can act as a carrier of the virus, is supported by Penberthy, Trinchera, Reindl, Sand, and Bang, who remarks: "It is self-evident that if the vagina is to be regarded as the chief port of entrance of the contagion, copulation must afford the most favourable opportunity of all for infection; as that is the only way in which a direct introduction of the virus into the uterus can be effected." Bang has proved that the disease can be set up in the mucous membrane of the uterus by an intravenous injection of pure cultures, in which case there occurs symptomatic fever without, however, morbid changes in any of the internal organs, except the uterus. Bang and others have shown that the bacilli, if placed in the vagina, can penetrate into the uterus; although they do not always do so. The disease seems to be identical in cows, mares, ewes and she-goats, and can be communicated from any one of these animals to any of the others.—Tr.]



## DYSENTERY.

General remarks—Dysentery of man—Dysentery of sucklings—Dysentery of adult animals—Bovine dysentery from coccidia.

**General Remarks.**—The recent investigations of Jensen on the dysentery of sucking calves, and those of Zschokke, Hess, and Guillebeau on that of full-grown cattle, have thrown some light on the nature of dysentery in our domestic animals. Dysentery in them may be divided into dysentery of sucklings and dysentery of full-grown animals. These two forms must be regarded as independent diseases. The dysentery of horses, dogs, sheep, and birds has, however, been but little studied. The question whether the dysentery of our domestic animals is etiologically identical with that of man, still remains unanswered.

**The Dysentery of Man** is essentially an infective, croupy, diphtheritic inflammation of the large intestine, with suppurating and hæmorrhagic infiltration of its mucosa and submucosa. Its virus, the nature of which is unknown, is probably not directly contagious. The infection is communicated by the evacuations of persons suffering from dysentery. The disease appears sporadically and endemically, and in our climate is on the whole benign, but is very malignant in the tropics. The symptoms consist of gradually increasing diarrhœa and tenesmus; colic; vomiting; mucous and subsequently purulent, and even bloody condition of the fæces; pains during micturition, and grave general disturbance. Complications sometimes set in, as for instance, abscess of the liver, a diseased condition of the articulations, inflammation of the serous membranes, perforative peritonitis, etc. Therapeutics consist chiefly in the administration of aperients (calomel and castor oil) and stimulants. [The most valuable drug in the treatment of human dysentery is undoubtedly ipecacuanha.—Tr.]

## THE DYSENTERY OF SUCKLINGS.

**Occurrence.**—The dysentery of sucklings chiefly occurs in calves; though it also affects pigs, lambs, foals, cats and dogs. Next to pyæmic polyarthritis, it is the most destructive disease peculiar to sucklings. For several years it has steadily increased in various provinces in Germany. Many farms have been so severely attacked, that the raising of young cattle has been rendered impossible. Röhl reports that, out of a total of 3,318 calves, 1,196 became infected, and 1,152 died (a mortality of 97 per cent.). In the year 1884, 55 per cent. of all the affected animals died in some parts of Austria. It attacks calves almost

exclusively during the first 3 days of life. The animals often become affected directly after birth, even before they have taken any milk, a fact which goes far to prove that the mother's milk does not participate in the production of the disease. A further proof of the non-participation of the mother's milk in the transmission of the disease, is the practical observation, that dysentery also appears when milk from non-affected sheds or boiled milk is given, and even when substitutes for milk are employed.

**Etiology.**—The cause of the dysentery of calves is, according to Jensen, an oval bacterium which appears singly, in pairs, or in the form of chains, and is either a pathogenic variety of the bacterium coli commune or identical with it. Jensen considers that the disease is not due to a specific contagium, but to a pathogenic variety of the bacterium coli commune, which is normally present in the intestine. The usually harmless bacterium coli commune may become pathogenic, and may penetrate, as the exciter of the disease, into the intestinal mucous membrane, when the newly-born calf becomes exposed to debilitating influences, such as chill, errors of diet, etc. Cultivations of the intestinal bacterium which has become pathogenic, also produce dysentery when given along with the food to healthy calves. Intraperitoneal inoculation produces in guinea-pigs a fatal, very acute, sero-fibrinous peritonitis. The direct transmission of the disease by experimental feeding with diarrhoeic fæces has proved unsuccessful (Gutmann). On the other hand, it seems that the disease can be transmitted from one species of animal to another, as for instance, from calves to lambs and pigs (Kotelmann). It has been frequently observed that epizootic abortion and dysentery of calves occurred at the same time. It was also supposed that the fœtus might become affected by infective matter which had penetrated the vagina of the pregnant cow, and that this theory explained the special frequency of the disease during the first days of life. Jensen considers both suppositions to be incorrect. Schütz has confirmed the results of Jensen's bacteriological researches. According to him, the intestine is the source of the infection. The contagium is disseminated throughout the shed by the fæces of the infected calves, and gets into the bodies of healthy calves by means of intermediate bearers, such as farm servants and the dung-soiled teats of cows.

**Anatomy.**—There is nothing specially characteristic about the anatomical changes. The intestinal mucous membrane is covered with mucus and pus, which contain a great quantity of bacteria. The epithelium of the mucous membrane is absent in some places; and the mucous membrane itself is loosened and swollen, and sometimes shows a superficial diphtheria of Peyer's patches. In very severe cases, the contents of the intestine are more or less bloody (hæmorrhagic intestinal inflammation). Ulceration does not occur, apparently on account of the short duration of the disease. The mucous membrane of the abomasum, especially on the top of the folds and in the region of the pylorus, is hyperæmic; it contains ecchymoses, is cedematously swollen, and is frequently in a state of maceration. The contents of the abomasum generally consist of caseous coagula, and has a normally acid reaction. We have sometimes found in it traces of blood. The excessive general anæmia of the cadaver, especially of the internal organs, such as the liver and the kidneys, and the parenchymatous changes of the liver, muscular system, and heart, are very striking. A lobular broncho-pneumonia may be found as a consequence of drenches.

**Symptoms.**—The symptoms of the dysentery of sucklings are exactly the same in the different species of animals. The *calf*, during the first few days after birth, loses more or less its desire to suck; it suffers from pappy diarrhœa and tenesmus, is restless and lows. Later on, the excrements, which at first were yellow, become whitish and very thin; they assume a mucilaginous condition, are mixed with coagulated milk (so-called "white" dysentery) and frequently with blood, and are then very foetid. Finally, the fæces are passed involuntarily; there is excessive weakness; and the animal lies down continuously, suffers from convulsions, and allows the saliva to flow from the mouth. Death often takes place within 24 hours, although the average period of the duration of the disease is from 1 to 3 days. The mortality varies from 80 to 100 per cent. Very frequently all the calves of a shed die one after the other. The survivors generally remain sickly for a very long time.

*Lambs*, also, give up sucking; they become depressed and very weak; and, suffering from diarrhœa accompanied by tenesmus, they pass thin, mucilaginous, foetid fæces. At first the temperature may rise to 41.5° C., but rapidly falls towards the

end (Nikolski); respiration is accelerated, and saliva and mucus flow from the mouth. As with calves, the duration of the disease varies from 1 to 3 days, but death sometimes ensues in a few hours. The disease in lambs is also specially prevalent during the first 3 days of life. According to an observation reported by Nikolski, 30 per cent. of the infected lambs become ill on the first day after birth, 40 per cent. on the second, 25 per cent. on the third, and only 5 per cent. subsequently. He also mentions that, out of a herd of 12,000 sheep, 50 per cent. of the lambs died from this disease.

Dysentery is also found in *foals* during the first 3 days after birth (Mazoux), and sometimes later on. The animal becomes depressed and restless; the frequently-passed fæces are foetid, or thin and mucilaginous; the breath and the exhalations from the skin often have a very bad smell; the eyes are sunken; the animal becomes very weak, very thirsty, and greatly tucked up in the flanks; and sometimes there is a cutaneous eruption, which spreads over the whole body, and is particularly well-marked about the anus.

**Diagnosis.**—We diagnose dysentery from the fact of its enzootic character, and from the presence of severe and rapidly fatal diarrhoea during the first few days of life. In sucklings, the disease is most readily mistaken for acute simple gastro-intestinal catarrh, due to errors of diet and defective condition of the milk of the mother, which affection of the alimentary canal does not, as a rule, become developed soon after birth. Also, its course is milder than that of dysentery.

**Therapeutics.**—The sick animals should be segregated, the infected sheds thoroughly disinfected, the udder most carefully purified, and the female organs washed out with a disinfecting fluid, both before and after parturition. If the disease is enzootic in a shed, pregnant cows should be put a few days before calving into a fresh and clean shed. The observance of these precautions has proved to be far more effective than all the medicines that have been used against dysentery. It is advisable at the first appearance of the disease to give a mild laxative, such as castor oil for calves and lambs, and small doses of calomel (0.1 to 0.2 gramme *pro dosi*) for foals. Usually, rhubarb root (2 to 5 grammes for calves, and  $\frac{1}{2}$  to 1 gramme for lambs) and opium (1 to 2 grammes for calves, and 0.1 to 0.2 gramme for lambs) are given alone or combined. We can

recommend the following mixture : pulv. rhei radix 5 grammes ; pulv. opii 2 grammes ; and pulv. magnes. Carbon. 1 gramme ; to be given in one dose to a calf with 100 grammes of camomile tea, or 50 grammes of whisky. Instead of opium, we may give its tincture, 7 to 14 grammes for calves and foals, and 1 to 2 grammes for lambs. Opium and its tincture should be given in much larger doses than were formerly employed. The doses mentioned may be repeated (on an average 3 times with intervals of 6 hours) until the diarrhoea has ceased. Also tannin (1 to 2 grammes for calves, and 0.2 to 0.5 gramme for lambs) may be given either alone or combined with an equal quantity of salicylic acid, as for instance, 2 grammes of each in camomile tea, once or twice a day for calves. This combination is greatly esteemed by many persons. The following agents are also held in repute : nitrate of silver (1 tablespoonful of a  $\frac{1}{2}$  per cent. watery solution every 3 hours for calves), creolin (three doses daily of 1 gramme in water for calves), tea (doses of  $\frac{1}{4}$  litre for calves), resorcin, naphthalin, salol, salts of bismuth, etc. We may use with advantage mucilaginous demulcents, such as linseed tea, decoction of althæa, oatmeal gruel, raw eggs, etc., alone or combined with opium.

#### THE DYSENTERY OF ADULT ANIMALS.

**Occurrence.**—This disease seems to occur most frequently in cattle, and is more common in stall-fed animals than in those on grass. It often ceases when the animals are turned out on pasture. It may sometimes remain for several years in a shed, in which case it seems to gradually assume a milder character. It may, however, break out sporadically. Such factors as chill, errors of diet, over-exertion, damaged fodder and bad water, bad seasons and times of war, which were formerly supposed to be its causes, have only a predisposing influence.

**Anatomy.**—The mucous membrane of the large intestine is more or less hyperæmic and hæmorrhagic. It is often swollen ; in parts denuded of epithelium ; and covered with yellow diphtheritic deposits, which, later on, leave ulcers and scars. The contents of the intestine are foetid, and their colour varies from dirty-yellow to that of blood. The mucous membrane of the abomasum is also hæmorrhagically swollen, and the intestinal lymphatic glands, especially Peyer's patches, are inflammatorily infiltrated and ulcerated. In chronic cases, the

intestinal mucous membrane is sometimes thickened and of a slate colour ; the muscular coat of the intestine, hypertrophied ; and the intestinal serosa in the neighbourhood of the ulcers and scars, affected with adhesive inflammation.

**Symptoms.**—As a rule, the disease manifests itself suddenly by loss of appetite, high fever ( $40^{\circ}$  to  $41^{\circ}$  C.), rigors, debility, attacks of colic, and diarrhoea. Later on, the fæces become very thin, mucilaginous, frothy, discoloured, foetid, and even bloody. They are often mixed with undigested food, croupy membranes, and cast-off epithelium from the mucous membranes. At the commencement of the attack, tenesmus, and frequent, unsuccessful and severe straining to defecate are present, so that prolapse of the rectum may take place. Later on, however, the fæces flow involuntarily from the paralysed, gaping anus. The mucous membrane of the rectum is excessively congested and swollen. The animals evince pain on palpation of the hind-quarters. On pressing the right flank with the hand, we may sometimes feel well-marked fluctuation of the intestines, which are filled with fluid (Albrecht). Weakness and emaciation increase ; the internal temperature falls below normal ; and death often ensues in as short a period as 1 or 2 days, but usually not until 2 to 4 days. Mild cases recover in 2 or 3 weeks. The chronic course of cases which continue for weeks or months is probably due to the presence of intestinal ulcers. The prognosis, even in grown-up animals, is always doubtful. The mortality is about 50 per cent.

**Differential Diagnosis.**—In full-grown cattle, it is difficult, and may be impossible, to differentiate dysentery from toxic, mycotic, and enzootic intestinal inflammation. The results of *post mortem* examination of cases of dysentery sometimes closely resemble those of rinderpest. It is important to remember that in dysentery, only the intestinal canal is affected, and that a direct infection can never be proved. A distinction between dysentery due to a toxic or mycotic inflammation of the intestines is not always possible.

**Treatment.**—Besides mucilaginous agents, such as linseed tea and a decoction of althæa, we give opium (10 to 25 grammes) and astringents, such as sulphate of iron, tannic acid, sugar of lead, and nitrate of silver. Coarse fodder should be avoided.

**Bovine Dysentery from Coccidia.**—Zschokke, Hess, and Guille-

beau have closely studied a form of dysentery which occurs among cattle in Switzerland, and to which they have given the names of red dysentery and dysentaria hæmorrhagica coccidiosa. It attacks young animals, especially those on grass, enzootically; and older ones, sporadically. On a pasture, or in a cowshed, it spreads from one animal to another in rapid succession, until several or all of the cattle become affected. It appears chiefly when the animals are fed on grass, and when they are turned out to pasture, and consequently in summer and autumn. It is supposed that the coccidia, which are specific infective protozoa, are received into the system along with the food and water, and that they immigrate into the epithelium of the intestinal mucous membrane, and set up severe inflammation in it. The disease can be produced experimentally in cattle by sporulating coccidia.

The period of incubation is about 3 weeks. In slight cases, the general health is at first but little disturbed. In severe cases there are rigors, and the temperature rises within the first 24 hours to 41° C. In from 2 to 6 days, the animal evinces great weakness of the hind-quarters, has difficulty in getting up, and persistently lies down; the upper eyelids become œdematously swollen, and the eyes sunken. The appetite becomes suppressed after from 4 to 6 days, although the thirst is great. Sometimes, even during the first 24 hours, the patient becomes very restless, has slight colic, and the movements of the flanks are decreased or entirely stopped. At first, the peristaltic action of the intestines is vigorous, but becomes arrested later on. In the early stages of the attack, defecation is normal, except that it is more frequent than usual. The fæces are often mixed with blood coagula the size of a pea, bean, or even that of a child's fist. In some rare cases there is at first severe, bloodless diarrhœa, combined with violent straining, in consequence of which prolapsus recti appears 2 or 3 days later, and numerous small blood coagula are sometimes found in the now thin, sanious, or entirely bloody fæces. It may happen that in exceptional and severe cases, violent straining, and severe dysenteric diarrhœa are present from the commencement. The intestinal evacuations always contain coccidia. In a few instances we may find croupy membranes and a highly purulent condition of the fæces.

The course of the disease is generally acute; seldom peracute. In large herds this disease often rages for several months. In mild cases, with a normal condition of the fæces, recovery may take place in a week; or in 2 or 3 weeks if only simple bloody diarrhœa is present. In severe cases with violent initial intestinal hæmorrhage, the animal may die within 24 hours. Relapses frequently occur; but are not dangerous.

By *post mortem* examination the small intestine is seen to have a dark brown-red colour, with dark-blood contents. The mucous membrane of the rectum shows thin bloody deposits, swelling, and petechiæ. The fæces contain numerous coccidia.

The treatment consists in feeding with dry food, as a prophylactic measure; in keeping the sheds dry and clean; and in giving sulphur, creolin, and lysol combined with mucilage or milk. As it is supposed that the infection is principally transmitted by drinking water; it is advisable to guard the wells with masonry, and to preserve the rain water as pure as possible

## ERYSIPELATOUS DISEASES OF PIGS.

*Swine Erysipelas, Contagious Pneumonia of the Pig, and Swine Fever.*

**General Remarks on the Erysipelatous Diseases of Pigs.**—Little was known concerning the respective differences between the epizootic diseases of swine, until the investigations of Eggeling, Löffler, Schütz, Lydtin, Schottelius, Bang, and others, lately threw light on their respective etiology, symptoms, and anatomical changes. Even at the commencement of the eighties, almost all the epizootics of swine were included in the collective name of "swine erysipelas." The researches of the last 10 years, however, enable us to divide them into the following independent diseases:—

1. *Swine erysipelas*, which is a specific septicæmia produced by a minute bacillus that manifests itself anatomically by hæmorrhagic gastro-intestinal inflammation, inflammation of the kidneys, swelling of the spleen, and parenchymatous inflammation of the liver, heart, and muscular system.

2. *Contagious Pneumonia of the Pig or Swine Fever* [see p. 85], which is caused by an ovoid bacterium, and occurs in two principal varieties, namely: (a) The pulmonary form, which appears as an infective inflammation of the lungs and pleuræ, with a tendency to necrosis and caseation of the lungs; and (b) the intestinal form, which consists of intestinal diphtheria with caseous necrosis of the intestinal mucous membrane.

**History.**—The name of swine erysipelas (splenic erysipelas, swine typhus, contagious pneumonia of the pig, St. Antony's fire, and petechial fever) was formerly given to several porcine diseases which externally resemble each other, only by the fact that they respectively produce redness of the skin, which is a symptom as common to entirely different diseases in swine, as is colic or hæmaturia. In former times, the following diseases were, evidently, frequently included under the term, swine erysipelas: urticaria; anthrax; true erysipelatous inflammations of the skin; septicæmic diseases; poisoning; perhaps, also, diseases caused by worms, such as that due to *strongylus paradoxus*; heat apoplexy; suffocation; various internal acute diseases which are accompanied by redness of the skin, as for



instance, inflammation of the lungs; and probably several infective diseases, the pathology of which is not yet fully known. Consequently, there were great differences of opinion on the nature of swine measles. At the end of the fifties, Nicklas and others drew attention to the fact that swine erysipelas (measles) was not anthrax; because, as general experience proved, the eating of the flesh of measly pigs produced no ill consequences to man. This anthrax theory became absolutely untenable in the year 1865 by the researches of Brauell, who discovered the bacillus anthracis. He showed that pigs were immune to inoculations of anthrax blood; and that inoculations with the blood of measly pigs did not produce anthrax. Besides, the bacilli of anthrax were never found in the blood of pigs suffering from swine erysipelas. Also, Harms, in 1869, described swine erysipelas as an independent infective disease which consisted essentially in a severe affection of the gastro-intestinal mucous membrane, with parenchymatous changes in the liver, kidneys, muscles, and lymph glands, and which did not attack the lungs.

In 1882, Eggeling distinguished the following forms of swine erysipelas: (1) *Sporadic erysipelas of the head*, which is an infective disease due to wounds, with frequent gangrenous desquamation of inflamed portions of the skin, and which resembles human erysipelas of the head. (2) *Sporadic and benign urticaria*. (3) *Epizootic erysipelas*, which may be compared to scarlet fever of man. It is an acute exanthema which may be transmitted by inoculation, and which is distinguished by constitutional disturbance; copper-red, and spotted discoloration of the skin of the intermaxillary space, lower portion of the chest, space between the fore-legs, abdomen, and inner surface of the thighs; inflammation of the mucous membranes of the respiratory organs; oedema of the lungs; and changes in the liver and kidneys. (4) *Contagious pneumonia of the pig*, which is a form of septicæmia, and which is the most frequent and most dangerous porcine epizootic. Its symptoms are severe constitutional disturbance, weakness, high fever, and a uniform redness of the skin, which rapidly spreads from the posterior parts of the abdomen over the whole body. The most important anatomical change observed in the disease is a hæmorrhagic gastro-intestinal inflammation, which is accompanied by great swelling of the mesenteric glands.

Löffler, in 1882, divided swine erysipelas into true swine erysipelas and contagious pneumonia of the pig. Here, Löffler's

classification is the reverse of Eggeling's; for his erysipelas is identical with Eggeling's contagious pneumonia; and his contagious pneumonia is probably included in Eggeling's erysipelas. The names introduced by Löffler have, however, been retained. His bacteriological researches have further furnished the following results. His erysipelas is caused by a very minute bacillus which closely resembles Koch's bacillus of the septicæmia of mice. It invariably kills mice by inoculation, and either kills rabbits, or makes them immune against the disease after they recover from the erysipelatos skin inflammation which is frequently produced by it. But contagious pneumonia of the pig is produced by small ovoid bacteria which greatly resemble Gaffky's bacteria of the septicæmia of rabbits. Inoculations of their pure cultures kill mice, guinea-pigs, birds, and pigs. Schütz found and described, in 1885, Löffler's erysipelas bacilli at first in the spleen of a pig which was suffering from measles and which had been brought from Baden; and later on, in pigs near Berlin. In the same year, Schottelius made similar observations in Baden. The nature of swine erysipelas has been fairly-well elucidated by the protective inoculations and researches of Lydtin, Schottelius, and Schütz. Pasteur, Cornevin, and Jensen have also studied this subject.

About the time when the differentiation between erysipelas and contagious pneumonia was made in Germany, a very infectious porcine epizootic, which was known as hog cholera, swine fever, and infectious pneumo-enteritis, broke out in America and England, and later on in Sweden, Denmark, and France. Its principal phenomena were intestinal diphtheria and pneumonic changes. Klein, Billings, Salmon, Bang, Schütz, and others examined it bacteriologically, with such contradictory results that the pathology of the disease still remains unsettled. The clinical and anatomical experiences, however, force on us the conclusion that the American swine fever does not differ, as was formerly thought, from German contagious pneumonia of the pig.

This subject is further discussed on page 85 *et seq.*

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#### SWINE ERYSIPELAS (*Rouget du Porc*).

**Bacteriology.**—According to the researches of Löffler, Schütz, and Schottelius, the bacteria of swine erysipelas are slender,

minute rods from 0.6 to 0.8  $\mu$  in length, and can be seen with exactness only when they are very highly magnified (oil immersion lens and Abbé's illumination apparatus). These bacilli are found everywhere in the capillaries of affected pigs between the red blood corpuscles, and frequently in the leucocytes. They are most numerous in the spleen, kidneys, and lymph glands. As we have already said, they closely resemble Koch's bacilli of the septicæmia of mice. Schütz, Kitt, Jensen, Lorenz and others state that they are identical with them. They thrive best in slightly alkaline extracts of the flesh of horses, oxen and pigs, in blood serum and the aqueous humour of the eye at a temperature of 36° C. Even at a temperature of 15° C. they grow freely. On the other hand, they do not increase on potatoes, infusions of plants, or in broth made from the flesh of pigs which have passed through the disease (Schottelius). These bacilli are, on the whole, anærobic, and consequently grow best under the surface of nutrient gelatine. Stab cultures in gelatine have the aspect of a fir tree or of a brush for cleaning glasses.\* These bacilli probably do not form spores. Moisture seems necessary for their preservation in the open air. According to Kitt, they resist putrefaction. The bacilli can be easily stained with Gram's solution.

According to the researches of Jäger, Petri and others, the bacilli of swine erysipelas are destroyed by slow drying, in from 50 to 80 hours; by water at 46° C. in 20 minutes; by water at from 55° to 70° C. in 5 minutes; by water at 90° C. in 2 minutes; by dry air at 46° C. in 3 hours and a half; and by air at from -3° C. to -8° C. in 13 days. They are speedily killed by quicklime (1 to 2), chloride of lime (1 to 100), a hot solution of caustic soda or of caustic potash (1 to 100), carbonate of soda (1 to 100), sulphate of iron (1 to 3), permanganate of potash (1 to 20), sulphate of copper (1 to 400), and by all the stronger antiseptics, such as corrosive sublimate and creolin. A concentrated solution of common salt, like that used for pickling meat, takes 26 days to kill the bacilli; and brine, 7 days. Ordinary boiling and roasting will not kill all the bacilli in pieces of meat exceeding one kilo in weight; but two hours and a half boiling (not roasting) will destroy them in smaller pieces of meat. Sharp pickling for not less than a month will kill all the bacilli in infected meat. On the other

\* In this brush, the bristles radiate round a stem, with which they form a angle of 90°.—Tr.

hand, they remain unchanged in ordinary salt meat for a month ; in pickled meat, for 6 months ; and in smoked ham, for 3 months. We can see from these experiments that the ordinary methods of preparing pork are not sufficient to kill the bacilli in meat. Saturated solutions of boric acid, tannic acid, arsenic, benzine, chloride of zinc, and carbolic acid in spirit, do not accomplish that end, even in 48 hours. The bacilli lose their virulence in sea water after 12 days, but remain alive in it for over 24 days.

Inoculation succeeds most readily in white mice, which die under symptoms of septicæmia of mice. Rabbits die in from 5 to 9 days, or become affected with a local erysipelatous dermatitis, after recovering from which they are found to be immune to further infection. Pigs, pigeons and white rats die from the inoculation with the characteristic symptoms of erysipelas, and manifest bacilli in all their organs. According to Cornevin and Kitt, the inoculation is negative in guinea-pigs, fowls, geese, ducks, ordinary rats, dogs, cats, mules, asses, horses, cattle and sheep, all of which must be considered to be immune to bacillar erysipelas.

**Pathogenesis.**—According to Pasteur, Schütz, Schottelius, Lydtin and Cornevin, the bacilli enter the body chiefly by the digestive canal. Jensen considers that they can also get into the system by means of even very minute wounds of the skin. Buchner's supposition that the bacilli can be absorbed from the inspired air by the lungs appears impossible ; for Cornevin has proved that they lose their virulence by being dried. They rapidly increase in number after gaining entrance into the blood, and especially directly after death. Their pathological effect depends on the excretion of a toxin, which chiefly acts on the nervous system, the muscular system, and on the cells of the parenchyma of the large glands. Only by this fact we are able to account for the paralysis, grave cerebral disturbance, and anatomical changes in the large glands.

As a rule, one attack confers immunity.

**Occurrence and Dissemination.**—Bacillar erysipelas is an infective disease which is widely spread and which occurs enzootically and even epizootically, probably in most of the countries of Europe. Its dissemination corresponds to that of the bacilli of erysipelas, which appear to thrive particularly well in stagnant water. For instance Löffler states that it is

constantly found in the Panke [a small stream that runs into the Spree at Berlin]. Like anthrax, erysipelas is often stationary. It was formerly restricted in Bavaria to the districts of the Danube, and was entirely unknown in Southern Bavaria (Kitt). Lydtin states that the disease tends to become enzootic chiefly in valleys and low-lying plains which have slow-flowing streams, and heavy, damp clay soil ; and that sandy and granite soils are comparatively free from it. It occurs chiefly during July, August and September, and sporadically during winter. Sultry summer heat and thunderstorms seem to promote the development of the infective matter. According to Lydtin, the majority of cases in Baden have been observed on small farms which had insanitary pig-sties ; in places where great numbers of pigs were kept ; and where distillers' wash was used as food. It is usually supposed that sties which are damp, ill-ventilated, and polluted with the fæces of pigs, are especially favourable to the development of the disease. The same remark applies to bad, damaged food, which formerly was wrongly regarded as the direct cause of swine erysipelas.

Pigs are least predisposed to the disease during the first months of life. Cases occur most frequently among animals of from 3 to 12 months old ; and rarely after the age of 3 years. Sucking-pigs do not contract the disease by drinking the milk of infected sows ; though Koubasoff states that the bacilli pass into the milk. It has long been known that the respective susceptibility of different breeds of pigs varies greatly. Thus, Hable found that in Austria, imported breeds become more readily affected than indigenous swine. Lydtin states that in Baden, English and half-bred English (Suffolk) pigs become earliest affected ; then Polish-China pigs, and that Yorkshire pigs and their cross-breeds were less susceptible. The common country pig was least susceptible of all ; for which reason it is not suitable for inoculation experiments.

Swine erysipelas is chiefly a miasmatic disease, and thus differs from swine fever, which, as a rule, can be spread only by direct infection. The bacilli of swine erysipelas, similar to those of anthrax, become further developed outside the animal body (ectogenous development). Also, as with anthrax, an indirect infection takes place principally by means of the water and soil of the permanently infected districts. Direct infection may occur, but only by the absorption of infected excrements or of infected flesh. Transmission by the air does not take place. Hence, healthy pigs which live close to infected sties, do not

take the disease, if they are prevented by partitions from coming into direct contact with the sick animals, even when the air is allowed to pass freely from one sty to the other. Consequently, we must look upon the contagium as fixed.

The disease is often spread by the consumption of the flesh of infected pigs; by the diseased refuse of slaughter-houses and kitchens; by the water in which the flesh or the utensils of butchers have been washed; and by watering with infected vessels. Lydtin states that in a certain village, swine erysipelas was spread along the banks of a brook, into which the waste water of the neighbouring inhabitants went. Cornevin points out that the disease may be spread by giving pigs the brine in which infected pork has been salted.

**Symptoms.**—After a period of incubation of at least 3 days, the disease usually begins suddenly and violently. The animal refuses its food; sometimes vomits or makes efforts to vomit; has a very high temperature (up to 43° C.); manifests severe nervous disturbance; is very weak, torpid, sleepy, and indifferent to its surroundings; and tries to hide itself under its bedding. The hind-quarters become weak and paralysed. Muscular spasms and grinding of the teeth are sometimes observed. At first there is constipation; the mucous membrane of the eyelids is of a dark-red or brown-red colour; and the eyelids are sometimes swollen. Usually, a couple of days after the first manifestations of the symptoms or even from the very commencement of the attack, spots appear on the thin parts of skin, such as the region of the navel, lower surface of the chest, perineum, inner surface of the thighs, ears and throat. These spots, which at first are bright red and about the size of a man's hand, become, later on, dark red or purple, and soon unite into large irregularly-shaped patches. As a rule, they are neither painful to the touch nor prominent, but sometimes show a slight inflammatory swelling. The skin of the red spots, especially of the ears, may suffer from an eruption of vesicles, and may even slough. This spotted redness of the skin may be very slight in severe cases, or may appear only immediately before, or even after death. At other times it may be entirely absent, or may become spread over the entire body. Diarrhoea also sets in, and the faeces become thin, mucilaginous, and sanious in a few instances. Towards the end, respiration becomes greatly accelerated, general cyanosis (oedema of the lungs) supervenes, and death takes place, usually on the third or fourth day of

the attack, with increased general weakness and considerable fall of temperature (down to  $37^{\circ}$  C. or less). When the disease is very severe, the animal may die in 24 hours. Sometimes the disease takes a week or longer to run its course.

**Different clinical forms of Epizootic Erysipelas.**—Jensen considers that this disease, instead of being uniform in its clinical aspect, manifests itself in the following forms, which differ from each other by well-marked peculiarities; and that there may sometimes be transition forms between the respective varieties of the complaint:—

1. **True erysipelas** is the usual form of erysipelas, and is the one we have just described.

2. **Swine urticaria** is said to be a mild form of erysipelas, the character of which is due partly to the difference in the manner of infection (infection of wounds of the skin), partly to the slight degree of virulence of the contagium, and to the temporary diminished susceptibility on the part of the animal. It is said that the bacilli of erysipelas have been found in cases of urticaria (Jensen and Lorenz). Consequently, these two authors maintain that swine urticaria, which was formerly described among cutaneous affections as an independent, benign, infective disease, must be included in erysipelas. We, however, agree with Schindelka that no valid reason exists for referring all cases of urticaria of pigs to erysipelas; because an eruption of urticaria, as in other domestic animals, may certainly occur independently of erysipelas. The observations of Jensen and Lorenz, provided that they will be confirmed by further researches, are very important; for they appear to show that, apart from ordinary urticaria, there is a symptomatic nettle-rash which may be regarded as a form of epizootic erysipelas. According to Jensen, the symptoms of this form are as follows: The disease, as a rule, begins rather suddenly, by the animal ceasing to feed, showing high internal temperature from  $41.5^{\circ}$  to  $42^{\circ}$  C. and even to  $42.8^{\circ}$  C., and being extremely thirsty. The fever increases during the following two days, and the patient becomes languid, stiff, and so weak that it appears as if paralysed. One or two days after the manifestation of the first symptoms of the disease, more or less round, raised spots appear all over the body, and especially on the loins, outer surface of the thighs, neck, and shoulders, which are parts of the body that in ordinary erysipelas usually suffer from no cutaneous changes. These spots increase rapidly in size and number, and become dark-red, violet, or more or less black. On the third or fourth day, the wheals are sharply defined, and are generally square or rhomboidal, but may be round or oblong. They are 3 to 6 cm. in diameter, or even larger; are raised above the healthy skin only a few millimetres as a rule; and often run together so as to form large irregular, angular spots. They are of a dark-red colour, and number from 50 to several hundreds. In very slight cases, we may find on the entire skin only a single, square-shaped red wheal. After the development of the wheals, the fever increases and the animal begins again to feed. In a few days, the wheals disappear with desquamation of the epidermis; or in severe cases, with sloughing of the skin and the

formation of a dark, leathery crust. The whole duration of the disease varies from 8 to 12 days. The mortality is very small; as the course of the disease is almost always mild. Chronic endocarditis is a rare sequela. Urticaria and the ordinary form of erysipelas often appear simultaneously, or one after the other, in the same drove of pigs. Inoculations with pure cultivations of the bacilli of urticaria and with portions of the tissues of infected animals have almost always produced a negative result in pigs (Bang and Jensen).

3. **Erysipelas without redness of the skin** (French, *rouget blanc*) is a very rare and a very acute form of erysipelas, from which the sufferer dies too quickly for the redness of the skin to become developed.

4. **Diffuse necrotic erysipelas of the skin** begins, according to Jensen, with fever and with the appearance of irregular, flat, red swellings, about the size of a man's hand, on the neck, back, sides, and joints of the shoulders. These swellings tend to become confluent, so that almost all the skin on the upper part and sides of the animal is swollen and reddened. The red gradually becomes deeper in colour, or may turn into deep violet or nearly black; and the attacked parts of the skin lose their sensitiveness and warmth, become hard, dry, and necrotic, and slough with the formation of a line of demarcation. The ears and tail often rot off. The resulting cicatricial tissue is firm, more or less like cartilage, and subsequently contracts so much that the affected part becomes deformed. Some of the sufferers die at a very early stage, and others linger on for 1 or 2 months. Many pigs are slaughtered on account of their having become permanently disfigured. The cutaneous necrosis varies in size from spots that are as large as a man's hand, to patches which may extend nearly over the entire body. Bacteriological examination shows that numerous erysipelas bacilli are present in the lymph spaces of the cutis directly under the epidermis.

5. **Endocarditis of erysipelas** has been carefully studied by Bang. It often begins acutely with the ordinary symptoms of erysipelas, and is followed by a latent period, during which the animal apparently recovers; but subsequently becomes affected with a well-marked, visible, cardiac affection, which generally lasts for a week or two. In a few cases, the pigs die from apoplexy. The symptoms are: loss of appetite, depression, persistent lying down, shortness of breath, slight cough, and redness of the skin, in varying intensity and extent; although not to such a high degree as in acute erysipelas. After this, we find palpitation of the heart, greatly accelerated action of that organ, and some times endocardial murmurs. The temperature is often increased and the animal dies with symptoms of cardiac paralysis. In a few cases, we may observe paralysis of the hind-quarters. On *post mortem* examination, the left side of the heart is usually found to be affected; the right, less frequently. The ostium is almost always plugged up, and the mitral valve is the most frequent seat of attack. The valves are swollen and thickened, and show verrucose vegetations with deposits of thrombi, in which large numbers of erysipelas bacilli can be found. According to Bang, endocarditis of erysipelas is not rare. For instance, out of 10 pigs which had passed through an acute attack of erysipelas, 9 died



of endocarditis within 3 months. The fact of slaughtering for food animals which have apparently, but not really, recovered from epizootic erysipelas, greatly furthers the spread of the disease. Bang therefore recommends that the segregation of pigs recovering from erysipelas should be continued after they have apparently become quite healthy; or that they should be slaughtered and the owners indemnified.

**6. Other chronic forms of Epizootic Erysipelas.**—Cornevin, Hess, and others have described chronic cases which have become developed from an acute attack. The aspect of chronic erysipelas differs essentially from that of the acute form. Although the appetite remains good after the acute attack has subsided, the animal becomes emaciated, the belly is drawn up, and there is weakness, diarrhoea, and shortness of breath. The patient suffers from a chronic state of ill-health, which may last several months, and which, it is said, may be followed by symptoms of so-called "scurvy" or "bristle-rot," such as hæmorrhages in the bulbs of the hair, tendency of the bristles to fall out, and bleeding and ulceration of the gums. It is said that, besides these symptoms, we may sometimes find in certain organs, especially with inoculated animals, local changes such as enteritis, endocarditis verrucosa, and arthritis fungosa, with resulting chronic lameness and deformity of the skeleton. It is also stated that redness of the skin can be observed at the beginning and during the exacerbations of almost all these chronic cases, and that it is an infallible sign of approaching death.

**Prognosis.**—The ordinary kind of epizootic erysipelas has a mortality of from 50 to 85 per cent. Lydtin states that the losses from it in Baden were between 50 and 75 per cent. It is stated that during 1873 in Denmark and Schleswig, 87 per cent. of all the pigs affected with erysipelas died. In any case the prognosis is always very unfavourable, as we may judge by the fact that the financial losses caused by epizootic erysipelas and contagious pneumonia in different countries far exceed those from every other animal disease. During 1891 in Württemberg, 21,500 pigs out of a total of 350,000 suffered from erysipelas (6 per cent.), with a loss of £22,000. Zschokke and Hess state that the yearly loss from it in Switzerland amounts to many thousands of pounds sterling. The value of the pigs which died of erysipelas in Brittany during 1881 was £120,000. In France during 1887, out of 19,000 infected pigs, more than 15,000 died; thus showing a loss of £20,000. The German Imperial Board of Health puts the yearly loss of the German Empire from swine erysipelas at £225,000.

The hopeful cases are those which live over 4 days. According to Jensen, the prognosis of the urticarial form is very favourable.

**Anatomical Changes.**—In *post mortem* examinations of cases of the ordinary form of epizootic erysipelas, we find signs of septicæmia without any well-marked morbid condition of separate organs. This septicæmia is a general infection which produces hæmorrhagic and diphtheritic gastro-enteritis, considerable swelling of the lymphatic system, hæmorrhagic or parenchymatous nephritis, acute swelling of the spleen, parenchymatous hepatitis, and myositis.

1. The *hæmorrhagic gastro-enteritis* consists at first of excessive inflammation of the mucous membrane of the stomach in the region of the fundus. The mucous membrane shows a dark-red discoloration, which is partly diffuse, partly in spots; suffers from cloudy swelling; often has eminences on its surface; is covered with a viscid layer of mucus; and may even have superficial scabs. The glands of the mucous membrane are inflamed (gastritis glandularis). The intestinal mucous membrane is swollen, especially on the top of the folds, narrow parts of the small intestine, and in the neighbourhood of Peyer's patches; is covered with reddish mucus; infiltrated with hæmorrhages; and sometimes shows superficial scabs. Less frequently, circumscribed parts of the mucous membrane of the cæcum and of the anterior part of the colon suffer from a diphtheritic affection.

2. The *solitary follicles and Peyer's patches* present, throughout, medullary swellings in the form of prominent raised patches of the size of a grain of millet to that of a lentil. Sometimes they are infiltrated with hæmorrhages and surrounded by a red ring. We very frequently notice ulceration and cicatrisation of the solitary and agminated follicles. The mesenteric glands become more swollen than the other glands of the body; are of a dark-red colour; and show softening. The surface of their section is dun-coloured with interspersed dark-red spots; and the paraglandular tissue is hyperæmic, and infiltrated with hæmorrhages.

3. The *hæmorrhagic nephritis* is distinguished by enlargement of the kidneys, and by the kidneys assuming a grey-red colour. The medullary layer is generally of a very dark red, and the cortical layer is infiltrated with blood points and widened out. In slight cases, we find only a parenchymatous inflammation (cloudy swelling) of the kidneys. Frequently, catarrhal nephritis occurs as a complication.

4. *Acute swelling of the spleen* arises in consequence of an acute, severe hyperæmia with great increase of the cellular

constituents of the spleen (new formation of splenic cells), in which case the spleen is enlarged, but not softened as in anthrax. It is, on the contrary, tense to the touch, as the capsule is considerably stretched. The pulp on the surface of a section is purple, moderately soft, and free from hæmorrhages.

5. *The parenchymatous hepatitis* consists of a cloudy swelling and enlargement of the liver, in which the surface of sections has a greyish-brown colour, and the acini are widened cut. The muscles are grey in colour, soft, flaccid, watery, shiny, are sometimes infiltrated with hæmorrhages, and look as if they had been boiled. The myocardium shows similar spotted changes, and subendocardial hæmorrhages.

In the abdominal cavity, thoracic cavity, and pericardium, we sometimes find small quantities of an orange-coloured clear fluid, which may be mixed with flaky coagula; and punctiform hæmorrhages under the serous membranes, especially on the auricles. We rarely meet with severe hæmorrhages in the brain, or in the dorsal and lumbar portions of the spinal cord. The congested parts of the skin are somewhat discoloured after death. The lungs remain unchanged, or at most exhibit a *post mortem* œdema. By microscopical examination, the bacilli are found everywhere in the body, especially in the spleen and kidneys, and to a less extent in the blood.

[English veterinary surgeons, as a rule, regard the occurrence of more or less luxuriant vegetations on the valves of the heart (chiefly the mitral) to be so frequent in this disease, as to be almost diagnostic. It would appear that this endocarditis is not nearly so common in Continental cases.—Tr.]

**Diagnosis.**—Apart from the demonstration of the bacilli, the diagnosis of bacillar erysipelas depends essentially on the presence of swelling of the spleen, gastro-enteritis, nephritis, affection of the lymphatics, and absence of inflammatory changes in the lungs. In doubtful cases, we recommend the inoculation of white mice or pigeons with the blood or with small pieces of the spleen under the skin of the back or on the chest. When erysipelas is present, inoculated mice become affected in 24 hours. They are depressed and arch their backs; the coat stands on end; the breathing is accelerated; and the mucous membrane of the eyes discharges a mucilaginous, glassy secretion. Death takes place on the fourth day after inoculation. Inoculated pigeons die in 3 or 4 days. The erysipelas bacilli can be easily found in the blood of the inocu-

lated animals. For more exact diagnosis, Johnne recommends the preparation of a stab culture in the following way. The spleen of a dead pig is first of all washed in a watery solution of corrosive sublimate, 1 to 1,000, and torn asunder in the middle; a platinum needle which has been sterilised by being made red-hot and allowed to cool, is introduced into the middle of the pulp of the spleen 4 or 5 cm. deep, and is then stuck about the same depth into the meat peptone-gelatine. The test-tube containing the nutritive gelatine is held with its mouth downwards, and the cotton-wool plug is only removed directly before the inoculation. After 5 or 6 punctures have been made with the platinum needle and after the cotton-wool plug has been replaced, the test-tubes are allowed to stand for about 4 or 5 days in the ordinary temperature of the room, after which time, the characteristic slender cultivations, which radiate outwards like a pine tree, or like a chimney-glass brush, form in the nutritive gelatine.

**Differential Diagnosis.**—The following are the principal diseases which have to be distinguished from epizootic erysipelas :—

1. *Contagious pneumonia of the pig* or *swine fever* differs essentially from epizootic erysipelas by its bacteriological results (ovoid bacteria), and by the peculiar changes in the lungs and intestines. According to Cadiot, the two diseases may also be differentiated by inoculation. If, after inoculating a pigeon and a guinea-pig, we find that the former dies, and the latter remains unharmed, we may conclude that the disease is erysipelas; and contagious pneumonia, if the reverse of this takes place.

2. *True cutaneous erysipelas*, which is an infective disease, and arises from wounds chiefly on the head, is characterised by an inflammatory, painful skin affection, with tendency to gangrene (sloughing of circumscribed pieces of skin, and of the tips of the ears), and is localised principally on the skin of the head.

3. *Heat apoplexy*, which occurs especially in very fat pigs during transport when the weather is hot, and which causes a purple discoloration of the skin on account of vascular engorgement. The *post mortem* appearances are essentially those of suffocation.

4. As a rule, *anthrax* occurs very rarely in pigs, and then it is nearly always localised as gloss anthrax, or as anthrax angina,

in which case, there is difficulty in swallowing, dyspnœa, and carbuncular swelling of the skin and of the intermaxillary space. The presence of the bacilli anthracis, which are 10 times larger than the bacilli of erysipelas, confirms the diagnosis of anthrax.

5. Erysipelas may be confused with *traumatic erythemata*, as for instance, blows inflicted on driven pigs, and injuries of the teats or udder of a sow caused by sucking-pigs.

**Therapeutics.**—The course of epizootic erysipelas is so rapid that medicines are generally of no avail. In former times it was the custom at the beginning of the attack to give an emetic, which, we are unanimously assured, had a good result. Berner reported in 1858 that he cured 75 per cent. of all his cases by this treatment, the favourable effect of which is to be ascribed to the removal of the infective matter. On similar grounds, when treatment is possible, we can strongly recommend the administration of large doses of calomel (3 to 5 grammes), in order to thoroughly disinfect the stomach and intestines by the perchloride of mercury that is formed from the calomel. Creolin is also considered to be an effective remedy at the beginning of the disease. Obel recommends that 2 or 3 liqueur glasses of spirit (whisky, for instance) should be given later on in a quart of milk.

*Prophylaxis* is much more important than treatment. In the first place, all the healthy animals must be separated from the infected ones (not *vice versâ*). The infected sties should be thoroughly disinfected, and particular attention should be paid to the destruction of the excrements. According to the investigations made by the German Imperial Board of Health, quicklime dissolved in water, hot caustic solutions, and chloride of lime are efficient disinfectants. Freshly-bought pigs should not be placed at once in the sties, but should be previously kept for several days under observation in separate compartments. The consideration of the manner in which the disease spreads, teaches that we should exercise the greatest caution in exporting the flesh or offal of pigs that have died from erysipelas, or in feeding pigs on it. The regular official inspection of meat should on no account be neglected. The introduction of legal measures against erysipelas, similar to those which have been in force for a long time in Denmark and England, and since 1886 in Switzerland and Austria, is urgently needed in other countries. For instance, the following precautionary

legal measures exist in Austria (law of the 10th April, 1886) : The bodies of animals that have died from the disease, or of those which have been slaughtered and which show internal changes, must be rendered innocuous ; the consumption of the flesh of animals which had suffered from erysipelas, is permitted only in infected places ; pigs which have been exposed to infection must be slaughtered ; and the transport of pigs among which the disease breaks out while on a journey, must be stopped at once, and the animals must be placed in quarantine.

### **Protective Inoculation according to Pasteur.—**

Pasteur's protective inoculation is at present the chief prophylactic means employed against epizootic erysipelas. The exact method of preparing the inoculation material has not been published. We only know that Pasteur's vaccine is obtained by cultivating the bacilli several times in succession in the bodies of rabbits, and later on in glasses containing nutritive fluid. The animals inoculated *à la* Pasteur become, according to him, only slightly ill, and acquire immunity to erysipelas. The method of inoculation is as follows : The pigs at the age of 9 to 16 weeks are at first inoculated in the subcutaneous connective tissue with a weak vaccine (*premier vaccin*), and 10 or 12 days afterwards with a stronger vaccine (*deuxième vaccin*). The material for each pig costs about twopence half-penny, and may be obtained in Germany from Pasteur's laboratory in Stuttgart. The following results of this inoculation method may be quoted :—

1. In *Baden*, inoculation experiments were carried out during the years 1885 to 1887, under Lydtin. In 1885, 237 pigs of different breeds were employed. They were from 9 to 19 weeks old ; weighed from 11 to 47 kilos, and were distributed in 15 stations over the whole country ; so as to give due consideration to the respective influences of climate, soil, farming, feeding, and absence or presence of the disease in localities. The inoculation was carried out by an assistant of Pasteur in the following way. The pig having been placed on its back with its hind-legs held asunder, about 0.1 c.c. of fluid contained in a Pravaz syringe, which had been supplied by Pasteur, was injected subcutaneously on the inner surface of the thigh. Of the 237 animals, 119 were inoculated and the remaining 118 were kept as control animals. The inoculation of the first lot was made at two different periods, at

first with a weak material (*premier vaccin*), and 12 days afterwards with a stronger material (*deuxième vaccin*). Twelve days subsequently, 60 out of the 119 "protected" animals, and 60 out of the 118 "control" animals, were subjected to the influence of the infection; some of them receiving injections of Pasteur's *virus fort*, and others being fed on the flesh of pigs which had died from erysipelas. The respective results of the protective inoculation, and of the transmission of the disease by the *virus fort* or by infected flesh, were as follows: (1) Out of the 119 "protected" animals, 80 per cent. reacted to the first inoculation by a rise of temperature; 15 per cent. showed all the symptoms of erysipelas; and 5.4 per cent. died from erysipelas. By the second inoculation of the remaining 113 pigs, 46 per cent. showed a considerable increase of temperature; 7 per cent. manifested well-marked erysipelas; but there was no case of death. (2) Out of the 118 "control" pigs, 62 per cent. became infected by contagium received from the inoculated animals after the first inoculation and 1 died of erysipelas. After the second inoculation, 36 per cent. became infected with a rise of temperature; 4 showed well-marked symptoms of erysipelas; and 1 died from erysipelas. The effects of the *virus fort* were as follows: (a) Out of the 60 "protected" animals there was a slight rise of temperature among 19, and 4 suffered from a mild attack of the disease. (b) Out of the "control" animals, 61 per cent. became seriously affected, and 40 per cent. of these showed well-marked erysipelas. In all, 26 per cent. of the "control" animals died of the disease. In Baden, 26 out of 462 inoculated pigs (5.6 per cent.) died in 1886; but in 1887, only 2 out of 152 inoculated pigs (1.3 per cent.).

We can see from these figures that Pasteur's protective inoculation renders the inoculated animals to a certain extent immune; but the risk of the inoculation is not insignificant; for, in the first place, the inoculated pigs infect the healthy, and may thus spread the disease. Besides, the adoption of this system of inoculation renders the animals seriously ill, and is attended by a mortality of about 4 per cent., which exceeds by far the total loss caused by epizootic erysipelas.

2. In *Switzerland*, Hess inoculated with Pasteur's vaccine 46 pigs, of which 11 became violently affected with erysipelas by the first inoculation; 3 of them had to be slaughtered; 6 remained sickly; and only 2 made a complete recovery. No symptom of the disease was apparent directly after the second

inoculation ; but, later on, 9 became gradually sickly, and signs of enteritis, endocarditis, and arthritis were found on *post mortem* examination. Consequently, out of 46 pigs only 26 remained healthy ; and 15 of the remaining 20 became chronically affected. Hess and Guillebeau assume that these untoward sequelæ were caused by Pasteur's vaccine having become contaminated. They state that their confidence in it was shaken. Hess found that the inoculation was very liable to cause the death of old and fat pigs from erysipelas ; and that the inoculation of pigs more than 4 months old was hazardous.

3. In *France*, according to Chamberland, 1,067 died out of 75,455 young pigs which were inoculated during seven years (1886 to 1892) ; thus showing a mortality of 1.45 per cent. It is stated that before the introduction of the inoculation, the mortality amounted to 20 per cent., and in some districts even from 60 to 80 per cent. of all pigs.

4. Dieudonné inoculated during 1885 in *Lorraine* 21 pigs, all of which remained healthy. He inoculated 283 pigs during 1886 with a loss of 2.1 per cent.

5. Jakobi of Obornik carried out protective inoculations in *Prussia*. He reports that the loss amounted to 50 per cent. with old pigs. On the other hand, there were only 14 deaths among 1,036 pigs which were under 16 weeks old and which were inoculated according to Pasteur's method during the years 1888 to 1894. Röder had a similar favourable experience in Saxony in 1894.

6. Hutyra states that out of 787,772 young pigs which were inoculated in *Hungary* during the years 1889-1893, only 7,345 died (0.95 per cent.) in consequence of the inoculation. From this he concludes that Pasteur's protective inoculations might be used with advantage wherever the cost of the inoculation, by the experience of former years, does not exceed the losses caused by this disease.

7. In 1893, in *Württemberg*, 10 out of 32 inoculated pigs died, and the remaining pigs became more or less sickly.

8. In *Holland*, 1,829 pigs were inoculated during 1890, and 407 pigs during 1891 ; the result being that 1.7 per cent. of them died in consequence of the inoculation ; 6.4 per cent. had their health more or less impaired, and the remainder experienced no ill effect.

If we weigh all these experiences, we shall see that it is not yet possible to form a positive opinion on the merits of Pasteur's



method of inoculation against epizootic erysipelas. The resolution adopted by the Berne International Veterinary Congress (1895), that protective inoculation is an indispensable means for combating swine erysipelas, does not appear to be entirely warranted by the facts of the case.

**Lorenz's Protective Inoculation Material** is of unknown composition, and nothing positive can be said respecting its value.

[In England, swine erysipelas is neither markedly contagious nor infectious, and does not appear to occur either as an epizootic or enzootic disease. In fact, it seldom attacks more than 1 or 2 pigs in a herd.—*Sidney Villar.*]

## CONTAGIOUS PNEUMONIA OF THE PIG, AND SWINE FEVER.

[Contagious pneumonia of the pig and swine fever are entirely different diseases. The disease described by Friedberger and Fröhner in this chapter, is, on the whole, contagious pneumonia of the pig; although the two diseases have been mixed up to some extent, in the following pages. According to McFadyean, "the organism of swine fever is a bacillus whose length varies from 1 to 2  $\mu$ , and whose average breadth is about .6  $\mu$ . The bacilli do not grow into filaments either in the body or in the ordinary artificial media, and one seldom or never finds more than two rods joined end to end. The bacilli have rounded extremities, and apparently do not sporulate. They are actively motile, and when examined in a hanging-drop preparation, their movements are seen to be partly rotatory and partly fish-like or serpentine. Only a feeble growth takes place in bouillon flasks exhausted of air. The bacilli are readily stained by simple aqueous solutions of the ordinary basic aniline dyes, but they lose the colour by the method of Gram. When stained with aqueous methyl-blue solution, the majority stain faintly and uniformly, but some show the polar method of staining." Swine fever is called hog cholera in America; *pneumo-entérite infectieuse* and *peste du porc* in France; and *Schweinepest*, in Germany. Contagious pneumonia of the pig is termed swine plague in America; *pasteurellose du porc*, in France; and *Schweineseuche*, in Germany. Contagious pneumonia of the pig is seldom, if ever, met with in England; although sporadic cases of pneumonia, apparently caused by a bacterium indistinguishable from the microbe of German *Schweineseuche*, are of frequent occurrence in this country. For recent information on these two diseases, readers would do well to consult Nocard and Leclainche's *Les Maladies Microbiennes des Animaux*, which is a book that is well up to date.—TR.]

**History.**—I. Very little was known respecting contagious pneumonia of the pig (swine fever) in Germany before 1882. There is no doubt that this disease had raged

from time to time epizootically before this date in Germany and Austria, without its nature having been recognised. In former times it seems to have been confused chiefly with tuberculosis; a remark that specially applies to the enzootic so-called "caseous inflammation of the intestines," which was described by Roloff and Bollinger in 1875 and 1876. It was only in 1882 that the nature of the disease was elucidated by the investigations of Eggeling and Löffler. In Germany, it is at present the most widely disseminated and the most dangerous porcine epizootic; although swine erysipelas has diminished in frequency. Its increased spread during the last few years, is probably due to the importation of infected animals from foreign countries.

2. In *England*, Klein was the first in 1878 to describe under the name of "infectious pneumo-enteritis" (hog-plague, pig-typoid, hog-cholera, and swine fever), a swine epizootic in which the lungs, serous membranes, and intestines are specially affected. The mortality of this epizootic disease, which appeared for the first time in a widely disseminated form in England during 1862, is stated to have often amounted to 75 per cent. During 1885 in England, the disease attacked 40,000 pigs, out of which 27,000 were killed and 10,000 died; and 35,000 pigs died during 1886. Klein distinguishes a benign and a malignant form, both of which he considers to be very infectious.

[In *England*, the Board of Agriculture received from Parliament on the 1st November, 1893, powers to order the slaughter of swine suffering from swine fever, and of swine which had been exposed to the contagion of this disease. The number of pigs which were slaughtered as belonging to these two classes were as follows:—17,774 in the year 1893; 56,296 in 1894; 69,931 in 1895; 79,586 in 1896; and 40,432, in 1897. Although some headway has been made against this plague, "it must be admitted," to quote the Annual Report (1896) of the Board of Agriculture, "that there is but little in the history of the more recent outbreaks to confirm the conclusion that the extinction of swine fever can be anticipated in the near future." The great difficulty in stamping-out swine fever is chiefly due to: (1) The great power of resisting external influences which the virus possesses. (2) The long period during which infected pigs may remain apparently healthy. (3) The difficulty of diagnosing the disease during life.—Tr.]

3. During 1877 and 1878, a generally disseminated contagious pneumonia of the pig raged for the first time in *North America*. Detmers in Chicago termed it "swine plague"; Law in Ithaca, "hog fever"; and the farmers, "hog-cholera." This disease, which is stated to have mad

its first appearance in America about 40 years ago, is said to be very contagious and to manifest itself during life by drooping of the head, coughing, loss of appetite, rapid emaciation, great weakness, peculiar colour of the fæces, diarrhoea, constipation, and the appearance of pink spots on the skin of the abdomen, chest, inner surface of the thighs, and ears. The principal changes found on *post mortem* examination are those of inflammation of the lungs. We also have enlargement of the lymph glands and mesenteric glands; pleuritis; ulcerating changes in the colon; pericarditis; peritonitis; and parenchymatous changes in the heart. We find microscopically in all the fluid parts of the body, bacteria of different kinds, one of which Detmers called "*bacillus suis*," and has stated that it is characteristic of the disease. The mortality amounts to 75 per cent. During 1884 and 1885 in Nebraska 400,000 pigs (a quarter of the entire number) died in each of these 2 years. The total loss in 1885 to the United States was estimated at 30,000,000 dollars. In Missouri alone, 200,000 pigs died; and in Indiana 400,000 (one-fifth of the entire number). Salmon regarded the mild form of this epizootic, as, at first, a bronchial catarrh; and the severer form, as a catarrhal pneumonia. He also called attention to the fact, that American hog-cholera could not be identical with the German bacillar erysipelas (known in France as "*rouget*"); chiefly because the experiments with Pasteur's erysipelas inoculations did not afford protection against hog-cholera. He pointed out that erysipelas was furthermore distinguished from hog-cholera by its shorter period of incubation (3 days as compared with 7 to 14 days); by its briefer duration (2 days as compared with 8 or 10 days); by its smaller mortality; by the immunity of guinea-pigs; by the susceptibility of pigeons; by the rarity of ulcers occurring in the intestinal canal (an almost constant lesion in hog-cholera); and especially by the assumed fact that the bacillus of erysipelas is essentially different in form and development from the bacillus of hog-cholera cultivated by Salmon. This observer also advanced the opinion that American swine plague is not an etiological unit; but that it consists of two entirely independent diseases, namely, hog-cholera (ulcerating enteritis), and swine plague (infectious pneumonia). Billings, on the other hand, has conclusively proved the unity of American swine plague, and has shown that its two forms, the pulmonary and the intestinal form, are produced by the same bacterium.

4. In 1887, American (or English) swine fever spread first to *Sweden*, and from thence to *Denmark*. Several young pigs died in the middle of September 1887 on the island of Amagar, from which place the epizootic spread to Copenhagen and the island of Zealand, where it was minutely investigated, especially by Bang and Schütz, and was identified as the English, *i.e.* the American, disease. From that period, the epizootic spread to the continent of *Europe*, and from time to time became widely disseminated, particularly in *Prussia*, *Hungary*, and *Servia*. It was also introduced into *France* (Marseilles epizootic). The clinical and anatomical results leave no doubt that the swine disease which was observed in England, America, Sweden and Denmark, and also in Germany, Austria, Servia and France, is one and the same disease.

**Bacteriology.**—The statements of those bacteriologists who consider that contagious pneumonia of the pig and swine fever are independent diseases, are not only contradictory for the most part, but, above all things, are opposed to clinical and anatomical experience. We shall now briefly state the respective and supposed specific results obtained by bacteriologists.

The contagium of **contagious pneumonia of the pig** is, according to Löffler and Schütz, an oval non-motile bacterium of  $1.2\ \mu$  length and from  $0.4$  to  $0.5\ \mu$  in breadth. It can be stained only at its ends, and increases by division of the middle unstained part. It closely resembles the bacterium of septicæmia of rabbits, and, according to some, is identical with it. It resembles to an equal degree the respective bacteria of chicken cholera and septicæmia hæmorrhagica. Stab-cultures in meat peptone-gelatine show along the track of the puncture, numerous white points which become confluent and form a dirty-grey layer with peripheral punctiform foci. Around the site of the puncture a dirty-white wall forms, and the gelatine does not liquefy. In cultivations in broth, the bacterium grows slowly at the bottom of the test-tube in the form of a connected flake, which, on the test-tube being shaken, rises up like a twisted pig-tail of hair. It grows on potatoes only when the surface of the potato is slightly alkaline. The cultivations appear iridescent on solid blood-serum. They cannot be stained with Gram's solution, and thus differ from the bacilli of erysipelas. Mice and rabbits die within 2 days after subcutaneous inoculations with pure cultures, and the oval bacteria are found both in the blood and in the tissue fluids. Guinea-pigs, pigeons, rats, and domestic fowl are infected only with difficulty. Two pigs died in 1 or 2 days after the inoculation, on the site of which a severe carbuncular, inflammatory œdema appeared. The oval bacteria were found in the bodies of these pigs, and their cultivations, on being inoculated into other animals, produced the same result. A pig which had been rendered immune to erysipelas, died of contagious pneumonia two and a half days after inoculation with cultivations of the oval bacteria, and a calf died with symptoms of septicæmia 6 hours after the subcutaneous inoculation. The bacteria are particularly

numerous in the necrotic foci of the lungs, in pleuritic exudates, and in the bronchial glands. They are also found in the blood and in the abdominal organs, especially in the spleen.

Frosch states that **swine fever** is produced by a longish, oval, motile bacterium which is from 1.2 to 1.5  $\mu$  long, and half as broad. It possesses a bright middle piece (girdled bacterium), and cannot be stained with Gram's solution. The bacteria, which occur singly or in pairs, are said to have rounded ends and to undergo changes according to their nutritive material, so that they can, for instance, assume the appearance of rods. Their characteristic automatic movement is said to be partly rotatory and partly in irregular curves, and to cease in old cultivations. It is stated that inoculations of small quantities of these bacteria kill mice in from 4 to 6 days, and rabbits in from 5 to 7 days. These bacteria are chiefly found in the spleen, blood, glandular organs, and muscular system of the body. Also, Bang states that swine fever has a specific bacterium. It is said that in the chronic form of the disease, mixed infections occur from the bacterium of contagious pneumonia of the pig (necrotic pneumonia), and from the bacillus of necrosis (intestinal necrosis).

**Etiology and Pathology.**—Contagious pneumonia of the pig is a very malignant and exceedingly infectious disease produced by the ovoid bacteria we have described. It chiefly attacks pigs which are only a few months old, and in this respect differs from erysipelas. The contagium is both fixed and volatile. It can enter the body by different routes, such as the lungs, intestines, and skin. In consequence of the volatility of the contagium, contagious pneumonia of the pig can be transmitted without direct contact. The respiratory organs are a frequent place of entrance. As we often find exclusively gastric changes without any implication of the lungs in contagious pneumonia; the bacteria of this disease then evidently effect an entrance into the blood through the alimentary canal, under which circumstances, the food and the feeding troughs seem to be the special vehicles for the infection. In exceptional cases the infection takes place through the skin, as is proved experimentally by inoculation. The place of entrance seems to affect the form assumed by the disease. Thus, we have a pulmonary form (inflammation of the lungs) originating from the lungs; and an intestinal form (enteritis), from the intestines. These two forms are frequently combined.\*

\* Sidney Villar considers that the term, pneumo-enteritis, often correctly indicates the seats of swine fever lesions. The experiments made at the R.V.C., London, and the reports of the officers of the Board of Agriculture (see *Journal of Comparative Pathology and Therapeutics*, 1896), suggest, however, that

The wide dissemination of the disease is caused by traffic in pigs. The disease is often introduced by apparently healthy pigs; because the period of incubation is comparatively long, and the virus, having great powers of resistance, evidently remains active for many weeks in the sties of dealers and in the trucks or cars used for transport. It is evident that there are various intermediate bearers, such as clothes, people, healthy pigs, etc. Colds caught during transport, sudden change of food, and excessive obesity may be regarded as predisposing causes. The period of incubation of contagious pneumonia of the pig is considerably longer than that of epizootic erysipelas; and averages from 8 to 14 days, with a maximum of 20 days, and a minimum, it is said, of 4 days.

**Symptoms.**—Contagious pneumonia of the pig does not run a uniform course. In the same drove of swine entirely different forms may appear simultaneously. The clinical results, especially, vary so much in particular outbreaks, that, being unable to furnish any typical description of the disease, we are reduced to dividing it into various sub-forms. Thus, anatomically, we have a pectoral form (inflammation of the lungs), an intestinal form (enteritis), and an exanthematic form (affection of the skin). For practical purposes it would seem to us better to divide it clinically into a peracute, an acute, and a chronic form.

1. The *peracute form* (Rust and Schindelka) of contagious pneumonia of the pig is so severe—death ensuing in from 3 to 10 hours—that it can seldom be studied. It consists of a highly feverish hæmorrhagic inflammation of the lungs, or of pure septicæmia. The animal suddenly becomes extremely ill, after it has been feeding with a good appetite only a short time previously. It suffers from rigors, staggers, and falls down; the temperature in the rectum rises to from  $40.5^{\circ}$  to  $42^{\circ}$  C.; the pulse is very frequent and irregular; the beating of the heart is almost imperceptible; respiration is difficult and accelerated; tracheal râles are present; there is a frequent moist cough; the expired air is very warm; the upper surface of the body feels as hot as fire to the touch;

pneumonia in cases of swine fever in England is very rare. McFadyean states that in the great majority of cases of pneumonia occurring in pigs suffering from swine fever, the lung lesions are ascribable, not to the bacillus of swine fever, but to an organism indistinguishable from the German *Schweineseuche* bacterium. —Tr.

the sufferer, when taken hold of, offers but little resistance; the grunting is faint and muffled; and we may sometimes observe a mucous discharge and hæmorrhage from the nose, and red spots on the skin about the ears, on the neck, and on the sides of the chest. The posterior part of the body is somewhat tympanitic. Pressure on the region of the stomach causes pain, and the animal sometimes shows an inclination to vomit. The peracute form may pass into the acute form.

2. The *acute form* appears as a pneumo-intestinal inflammation which is sometimes complicated with various kinds of exanthemata of the skin. The duration of this form of the disease, which is the one most frequently observed, varies from a few days to 2 or 3 weeks. There is a moderate fever (from  $39.5^{\circ}$  to  $40.5^{\circ}$  C.); short, dry, painful, and spasmodic cough, often lasting for a quarter of an hour, with attacks of suffocation; muco-purulent discharge from the nose; heavy pumping and panting breathing; cyanosis of the mucous membranes of the head; and dull percussion sound of the thoracic walls. Auscultation proves the existence of rhonchi and suppressed respiratory murmurs. The animal, which at first is sometimes somewhat excited, soon manifests great depression and weakness, and more or less loss of appetite; and may have difficulty in swallowing solid food. Constipation is generally present at the beginning of the disease, and often changes, later on, into diarrhœa, which may, however, be present from the commencement, if the digestive apparatus is the chief seat of the disease. In grave cases, diphtheritic ulcers form on the tongue, gums, buccal membrane, palate, and tonsils. The eyes are sunken, and there is sometimes conjunctivitis, keratitis, discharge from the eyes, and swelling and adhesion of the eyelids. In many cases the skin shows no striking changes; but in others we may find diffuse or spotted redness, vesicles and wheals with violent pruritus, and finally crusts which frequently cover the whole body, and which vary in size from a pea to a sixpence. According to Schindelka, three different forms of cutaneous changes occur in swine fever, namely: (a) A spotted exanthema on the region of the anus, lower surface of the body, and inner and outer surface of the thighs, which lasts only for 3 or 4 days, and takes the form of red spots the size of a lentil. Vesicles, and finally circular, brown-yellow crusts appear on these spots. (b) A pink uniform exanthema which is not sharply defined and shows a red rich discoloration of the skin. It begins

at the anus and spreads in 3 days over the lower surface of the body. It becomes pale from the fourth day, on account of the epidermis desquamating in small scabs. (c) An attack of urticaria with the formation of vesicles (*urticaria bullosa*), in which case there is an eruption of very rapidly crowding wheals, upon which vesicles, pustules, and crusts quickly form.

3. The *chronic form* of contagious pneumonia of the pig develops from the two other forms which we have just described. Its course lasts on an average from 4 to 8 weeks; but may sometimes be prolonged for several months. According to its localisation, the symptoms consist either of a chronic affection of the lungs, or of the intestines. The former assumes the character of pulmonary consumption with persistent dyspnoea, chronic cough, and great and continually increasing emaciation. The latter manifests itself particularly by the presence of fetid, green or greenish-yellow diarrhoea, which lasts until the animal dies, or which may change into constipation. The patient loses condition, especially about the hind-quarters; becomes anæmic; and shows great weakness and symptoms of paralysis. Palpation of the abdomen is painful, and nodular tumours are sometimes felt in it. In both these forms of the disease, the skin is frequently covered to a considerable extent with scabs, and the eyelids are closed up by crusts. Pulmonary and intestinal symptoms are often simultaneously present in the same animal.

**Prognosis** is unfavourable in all three forms. The peracute variety almost always ends in death. Recoveries are rare in the acute form, which either has a fatal termination, or passes into the generally incurable chronic form.

**Anatomy.**—There is as great variety in the anatomical changes of contagious pneumonia of the pig, as in the symptoms. The *post mortem* appearances are extremely varied, according to the localisation of the disease, and especially according to its course. Here, also, we can give no typical description, so have to content ourselves with detailing the different stages of the organic changes.

1. The *lungs* show specific changes in the pulmonary form. The appearance of multiple gangrenous pneumonia is particularly characteristic. We find in the lungs, and particularly in the anterior lobes, and at the root of the lungs, hard hepatised



spots of different sizes and of a very dark-red or grey-red colour on section. These spots show on their cloudy, slightly granulated section surface, numerous yellow or grey-yellow [buff-coloured], dry, caseous, circumscribed foci, which in peracute cases are punctiform and about the size of grains of oatmeal. In acute and chronic cases they vary in size from a pin's head to a hen's egg, or may be even larger, and are then sharply defined from the other lung tissue by a red, inflammatory line of demarcation, and later on by a firm capsule of connective tissue. These yellow deposits are dead portions of lung tissue formed in a manner exactly similar to that which takes place in equine contagious pleuro-pneumonia, in consequence of a multiple inflammation of the lungs with hæmorrhagic exudation and subsequent discoloration. A large area of necrotic lung tissue may be formed by the running-together of several of these foci. Sometimes no necrotic foci are present, in which case we have only a diffuse hæmorrhagic pneumonia. The larynx, trachea, and bronchi contain a frothy, serous, or sanious fluid. The bronchial glands and the mediastinal glands are greatly swollen, congested, and moist. They are also frequently hæmorrhagically infiltrated, and their capsule filled with blood.

2. The *pleuræ*, especially in cases of gangrenous pneumonia, sometimes show signs of acute, serous, or sero-fibrinous pleuritis; and in cases of some standing, signs of chronic adhesive pleuritis. In the former case, the more or less copious exudate is either clear and watery, or is opaque, mixed with flakes of fibrin, and is often found on both sides. Its extensive accumulation may lead to hydro-thorax with compression of the lungs. Under the coating of fibrin, the *pleuræ* appear cloudy, lustreless, and rough, like velvet. Adhesion of the visceral and parietal layers of the *pleuræ* frequently takes place as a consequence of chronic pleuritis, in which case, we often find that almost the entire surface of the lungs has become adherent to and united with the costal *pleuræ*.

3. The *pericardium* generally contains a considerable quantity of opaque or bloody fluid. In some epizootics, a fibrinous pericarditis, which occasionally leads to adhesion of the pericardium, constantly occurs. The muscular substance of the heart is relaxed, and is of the colour of untanned leather.

4. In peracute and acute cases, the *mucous membrane of the stomach and intestines* frequently show signs of hæmorrhagic gastro-enteritis, such as, great congestion, swelling, the presence of ecchymoses and ulcers, and the fact that the fluid contents of

the intestines are thin and sanious. In chronic contagious pneumonia, the intestinal changes are very characteristic, and consist essentially in a necrotic, caseous, intestinal inflammation, which specially involves Peyer's patches. A very characteristic *post mortem* appearance of the chronic intestinal form of contagious pneumonia, is the presence of more or less numerous yellow or grey-yellow [buff-coloured], cloudy, circumscribed spots or nodules in the mucous membrane of the posterior part of the small intestine, cæcum, colon, and especially on the seat of Peyer's patches. These spots and nodules are somewhat prominent and are of different sizes. At other times, they are superficial and are only as big as the head of a pin or a lentil; or they take the form of dry, round nodules that are about the size of a pea or a button, and extend through the entire intestinal wall as far as the serosa, which shows circumscribed changes due to peritonitis. Besides these nodules, we find on the intestinal mucous membrane, numerous buff-coloured, cloudy spots (commencing diphtheritis), or ulcers which are of different sizes, depth and form, and which sometimes reach to the serosa.\* Extensive diphtheritic formation of scabs has also been observed on the mucous membrane of the mouth and intestine. The mesenteric glands and the portal lymph glands are swollen and hæmorrhagically infiltrated, and frequently undergo caseation in chronic cases. The liver is enlarged and its edges are rounded. The spleen, on the contrary, is, as a rule, more or less normal in size and condition in acute cases. In chronic cases, caseous foci are sometimes found in it.

5. The *peritoneum* frequently shows signs of having suffered from a fibrinous inflammation causing cloudy swelling, congestion, hæmorrhagic infiltration, and the formation of a velvety coating of fibrin on the surface of the peritoneum, and the effusion of a considerable quantity of yellow exudate mixed with flakes of fibrin. In chronic cases, we find adhesive peritonitis with adhesion of the intestines, especially of the intestinal loops. Hæmorrhagic nephritis is sometimes present.

6. In the peracute form, we sometimes find only general changes of septicæmia, without any particular local affection.

\* McFadyean states that in naturally occurring cases of swine fever, diffuse diphtheritic inflammation (superficial necrosis), or, more frequently, ulcers are to be found, with few exceptions, in the large intestine *post mortem*; and that in the very rare cases in which these lesions are absent, we may observe in their place intense congestion and punctiform hæmorrhages.—TR.

In old standing cases, the dead body is greatly emaciated and very anæmic. The skin is covered with firm crusts, the removal of which displays ulcers or commencing cicatrization. Numerous oval bacteria are to be found in the organs which were inflamed, or which had suffered from necrosis.

**Differential Diagnosis.**—The presence of the oval bacteria will decide doubtful cases. The following are the chief diseases with which contagious pneumonia of the pig may be confused.

1. *Epizootic erysipelas* may be easily mistaken for the eruptive form of contagious pneumonia, which may assume even the appearance of urticaria. The bacteriological and anatomical result will decide the question.

2. *Tuberculosis*. The chronic necrotic form of contagious pneumonia (swine fever) possesses great similarity to tuberculosis; because it also gives rise to caseous foci in the lungs and in the intestines. Apart from the difference in the respective bacteriological results, we have the fact that tuberculous deposits become calcareous in time, and are, also, of various ages, as a rule.

3. *Phthisis verminalis* has been confused in former times with contagious pneumonia of the pig, in cases in which pulmonary worms were found in the lungs of pigs suffering from this disease.

**Therapeutics.**—Medicines, as a rule, are of no use. *Prophylaxis* is all important in this disease, which can be successfully combated only by veterinary police measures, the chief of which are: compulsory notification of outbreaks of the disease; strict prohibition of removal; supervision of pig markets and transport of pigs; periodical examination of pigs in the possession of dealers; thorough disinfection of sties and cars used in transport; and rendering the dead bodies innocuous. As experience proves that the contagium in infected sties frequently survives careful disinfection; the entire drove, in such a case, should be slaughtered, and new sties, if possible, should be erected on another site.

The statements which Zschokke made at the International Veterinary Congress at Berne in 1895 essentially agree with our theory of the identity of contagious pneumonia of the pig and swine fever. He states that it is hardly possible to find any difference between the respective bacteria, which evidently belong to the same group and differ morphologically

only on account of differences in external influences (quantitative, not qualitative, difference). In any case, the two diseases cannot be separated pathologically, anatomically, or from a veterinary police point of view. Zschokke drew the following conclusion, which was almost unanimously accepted by the Congress: "The two diseases (contagious pneumonia of the pig and swine fever), from etiological and practical reasons, should, under a common name, be added to the diseases which ought to be combated by the State; their compulsory notification should be enforced; and they should be included in the catalogue of epizootics, and should be separated, in it, from bacillar erysipelas."

[In England the law does not take any account of swine-pneumonia or swine-erysipelas, because these diseases seldom or never occur in the epizootic form in Great Britain.

The Departmental Committee appointed in 1895 recognised the unity of swine fever, which they say "cannot arise under any conditions which exclude the specific virus; in other words, it is not a sporadic disease, but one of the true contagia." The Committee did not extend their experimental researches to swine erysipelas, which they "believe to be of rare occurrence in this country in the epizootic form. A considerable number of cases of the chronic form, with valvular disease of the heart, have been discovered during the *post mortem* examinations conducted by the veterinary officers of the Board. But when it has been possible to trace the history of the pigs from which the diseased hearts were taken, it has been ascertained that the affection had not manifested any tendency to spread by contagion."

The following extracts from the Report may be of interest:—

"Swine fever may be defined as a contagious and infectious disease of the pig associated with a necrotic and ulcerative condition of the mucous membrane of the intestine, the morbid condition being nearly always most marked in the large intestine.

"The disease of the lungs which occasionally accompanies the disease in the intestine is either collapse or pneumonia. It is necessary, however, to observe that in none of the experiments performed for the Committee was pneumonia produced either by inoculation with pure cultivations of the micro-organism, or by feeding with the natural material obtained from animals suffering from swine fever.

"In very acute cases of swine fever, the disease may prove fatal within 2 or 3 days, and then the only lesions present may be intense inflammation of the stomach or intestines, or of both; and in these exceptional instances a certain diagnosis cannot be made except by bacteriological methods. . . . The acute and rapidly progressive form of the disease is commonly met with. There are also, and always have been, many cases of the obscure or chronic form of the disease, in which the morbid changes go on slowly for many weeks or months, and finally attain an excessive state of development without being attended by any symptoms which are usually accepted as diagnostic of swine fever.

"Some very important information in regard to the obscure form of swine fever was obtained by the Committee from the examination of swine which had been isolated for a period of 2 months on infected

premises. At the end of that time they had been certified by a veterinary surgeon to be free from swine fever, and would in the ordinary course have been released. In several of these instances, instead of being released, the swine were, at the request of the Committee, slaughtered, and the organs sent for examination. In each set of specimens, characteristic lesions of swine fever were detected.

"It was found that animals placed in contact with the diseased swine, or in the sties which had been occupied by them, became affected with a similar type of slowly progressive disease. On *post mortem* examination of all the original cases, the remarkable feature was the great disproportion between the very advanced lesions in the digestive canal, and the slight symptoms of disease exhibited by the animals during life."

Referring to clinical observations on the progress of swine fever among pigs which had been infected by feeding, by inoculation, or by exposure to natural infection, the Committee state:—"It will be noted that, taking the whole of the results, they were remarkable and quite unexpected. The well-known and characteristic symptoms of swine fever were not developed at any time during the course of the disease, which, in some cases, assumed a very exaggerated form. There was no redness of skin apparent in any case, and in one instance only were there any indications of loss of power in the hind extremities. Diarrhœa occurred in a few cases and continued for a short time, but in other instances constipation was present for a few days. The appetite was variable in some of the animals, but altogether they consumed a fair quantity of food up to the time of death or slaughter. In nearly all the swine which were examined *post mortem*, very pronounced lesions of swine fever were detected. In some cases there were large intestinal ulcers partly or entirely healed, the animals having, in fact, recovered from the attack."

Alluding to the chronic form of swine fever in four pigs, which while in one pen, had been fed on the intestines of a pig which had suffered from this form, the Committee observed that "none of the ordinary symptoms of acute swine fever, *i.e.*, discoloration of skin, loss of motile power in the hind extremities, cough, and diarrhœa, appeared, but instead the indications which are now unhesitatingly accepted as evidence of the chronic or obscure form of the disease, were developed early in the experiment. After a few days the eyes became dull and slightly sunken, the upper lids had a peculiar appearance due to the loss of hair, exposing the skin, which was covered with desquamating cuticle. A sticky secretion was discharged from the edges of the lids, the skin of the forehead was wrinkled, the hair (bristles) was slightly elevated over the surface of the body, the tail was limp and often the tips of the ears were dropped forward, and the animal exhibited a generally dejected appearance. Constipation alternated with diarrhœa from time to time, but neither symptom was so marked as to excite special attention. The pigs ate a good quantity of food, but never improved in size or in condition; indeed the tendency in these animals, as in the other cases recorded, was in the contrary direction.

"The pigs usually attacked their rations with avidity when first placed before them, but often ceased to eat before finishing their meal, returning to the trough again after an interval of rest.

"On January 30th 1 of the 4 pigs was killed, and on *post mortem* examination well defined lesions of swine fever were detected. There were necrotic ulcers on the ileo-cæcal valve, and several large ulcers with necrotic centres in the colon, and the mesenteric glands were as usual highly congested. Other organs of the body showed no indications of disease.

"It was deemed expedient to test the remaining 3 pigs as to their power to transmit the disease by association, and accordingly 2 young pigs were put in the pen with them on February 5th.

"On February 14th both pigs presented the appearance which has lately come to be recognised as the 'swine fever expression.'

"With very slight and temporary alteration of symptoms the 2 lately introduced pigs and the 3 which had been fed on December 24th remained until February 22nd, when they were all slaughtered.

"On *post mortem* examination of the 3 fed pigs, lesions were detected very similar in character and extent to those which were found in the pig killed on January 30th, but in all of them the majority of the ulcers were undergoing the healing process, and the mucous membrane in several parts of the cæcum and colon showed quite complete cicatrization. It was evident that the animals were recovering.

"The lesions of swine fever in the 2 pigs which had been kept in contact with the 3 pigs fed since February 5th were well defined, but the ulcers were in one case very small and numerous in the colon, and most of them in both pigs were healing rapidly, and there is no doubt that the pigs would have recovered."—TR.]

#### CHOLERA AND PLAGUE OF BIRDS.

Chicken cholera and fowl typhoid—Cholera of canaries and ducks—Klein's fowl disease—Epizootic dysentery—Maladie du sommeil—*Vibrio cholera* of fowl—Mycosis of parrots.

**History.**—Next to diphtheritic inflammation of the mucous membrane, chicken cholera is the most important and most common disease of poultry. It is stated that it was known in ancient times, and was considered by some to be identical with the cholera or with the typhus of man; hence the name. Others regarded it as a form of anthrax. Lemaistre states that it was for the first time investigated with an attempt at accuracy in Lombardy during 1789, and that it raged in the East Indies in 1817. It has settled in France since 1825, and has caused during 1830 and 1850, and from 1860 to the present time, great losses in poultry. During the thirties it seems to have spread to Russia, Austria, and Bohemia, and during the last decades it has become common in nearly every European country. It

has been for a long time the subject of veterinary investigation, and the knowledge of its symptoms, anatomical results, and mode of infection, was so far advanced, that only its bacteriology was left for modern research. As early as 1851, Benjamin assumed that a contagium was the cause of the disease and observed that men and dogs could consume the flesh with impunity. Delafond and others proved that the disease could be transmitted to poultry and rabbits by the inoculation of infected blood, secretions, and parts of the dead body; that retro-inoculation was possible; and that the fæces were connected with the spread of the infection. Feeding experiments confirmed its contagiousness; and it was even then observed that the contagium lost its virulence when it became dry.

Modern investigations were inaugurated by a comprehensive work of Perroncito. It is very probable that Perroncito was the first to see the characteristic bacteria of fowl typhoid which Toussaint was the first to cultivate in neutral urine. Toussaint declared, later on, that these microbes were identical with the ordinary bacteria of septicæmia, against which supposition we have, besides other considerations, the fact that fowl typhoid cannot be produced by the inoculation of septic substances. Pasteur cultivated the fungus in sterilised chicken broth and first proved in 1880 that one attack conferred immunity. He also recommended protective inoculation. Thus, fowl typhoid was the first disease against which Pasteur used his method of protective inoculation, and was the starting point of his theory of protective inoculation. Kitt has been particularly prominent in recent bacteriological research. The name of *bacterium avicidum* has lately been given to this bacterium.

**Bacteriology.**—The contagium of fowl typhoid is an exceedingly small, ovoid, motile bacterium from 0.3 to 1  $\mu$  in length. These bacteria, which can be seen only when they are considerably magnified (immersion), are very short, biscuit-shaped rods of the form of the figure 8. They are constricted in the middle and translucent, or appear as oblong or circular globules. Under comparatively low powers, they can be seen only as minute double points. They stain well with aniline colours, but not with Gram's solution. The cultivations consist of small, hyaline, white dots, which appear in great numbers close to each other under the gelatine, and may attain the size of the head of a pin (colonies of fungi). Besides this, weak, dull-white layers with spherical dots form on the surface of the

gelatine. The fungus grows best at a temperature of from 30° to 40° C. on neutral chicken broth, which is mixed with gelatine or agar-agar; and also on a solution of meat pepton, solution of extract of meat and sugar, hard-boiled white of egg, and solid serum. The bacteria can be transmitted to all kinds of birds, rabbits, and white mice, by cutaneous, subcutaneous, and percutaneous [inunction] inoculation, and by feeding on blood, fæces, and parts of infected dead bodies. The inoculation kills in from 12 to 48 hours. In guinea-pigs, abscesses, which contain large numbers of the bacteria, form on the site of the inoculation (Pasteur). Only local abscesses form in inoculated sheep and horses (Kitt); and in man, when the contagium comes in contact with cutaneous wounds (Marchiafava and Celli). Kitt observed that an injection into the udder of a cow, set up catarrhal mastitis, and that the bacteria were found in the milk for a long time. Dogs and cats do not become infected by feeding on the bodies of poultry which have suffered from this disease; and apparently the consumption of the boiled flesh does not transmit it to mankind, although Zürn has reported one case which suggests caution.

The bacterium of fowl typhoid is one of the easily destructible contagia. It is killed by mere drying, by most of the disinfectants, absolutely by boiling water, and by diluted sulphuric and hydrochloric acid (1 to 500). Kitt has found that it dies when exposed for three-quarters of an hour to a heat of from 45° to 50° C.; that it is not killed by a temperature of -14° C. lasting for 14 hours; and that it remains virulent for 3 months when associated with other bacteria. According to Massa, cultures continue to be very virulent for 2 months. Hueppe and Kitt state that the bacteria of fowl cholera are identical with those of the septicæmia of rabbits.

**Occurrence and Dissemination.**—Fowl typhoid, as we have already said, attacks all kinds of birds, especially poultry, pigeons, peacocks, pheasants, parrots, and canaries. It frequently stops the breeding of poultry by destroying all the birds in a poultry establishment, and even entire breeds. The infection is communicated usually by the consumption of the fæces of infected birds along with the food or drinking water; and often by the feeding on the offal of infected birds which have been killed. It is generally introduced into a poultry yard by newly-purchased, infected birds; or by neighbouring birds, as for instance, pigeons which visit strange poultry yards. The



importation of foreign poultry is a very common means of spreading this disease. Thus, the cholera of fowl and that of geese were introduced into Prussia chiefly by Russian, Polish, and Silesian poultry. Bavaria and Southern Germany were infected principally by Italian poultry. The disease is often spread by poultry shows. According to Barthelémy, infection may take place even by feeding on the eggs of infected birds ; because some of the bacteria which are within the body of infected hen-birds, may penetrate into the eggs, as has been proved to occur in the case of mammalian fœti (guinea-pigs). It is possible that the contagium may also be absorbed by wounds in the skin. Buchner has shown by experiment that the bacteria, on being inhaled, can pass through the intact surface of the lungs ; thus giving rise to the disease.

**Symptoms.**—Fowl typhoid is characterised by the remarkable rapidity of its course, and by the extreme brevity of its period of incubation, which averages 24 hours, and has a maximum of 48 hours, and a minimum of 18 hours. In many cases death occurs apoplectically in such a sudden manner, that no signs of the disease are noticed during life. The birds are unexpectedly found dead in the morning, or they suddenly drop down dead off their perches. Hens have been known to die of this disease while sitting on their eggs.

In most cases, the disease lasts somewhat longer than we have just said ; but on an average not more than from 1 to 3 days, and sometimes for only a few hours. The bird loses its appetite ; is depressed and weak ; droops its wings ; blows out its crop ; curves its neck ; separates itself from its fellows ; its plumage becomes ruffled ; and there is a rise of internal temperature of from 1° to 3° C. We sometimes notice the discharge of frothy mucus from the beak, and vomiting. Diarrhœa, accompanied by violent thirst, sets in. The fæces are at first pappy and whitey-yellow, but later on they become mucilaginous, watery, green, and fœtid, and consequently the parts near the opening of the cloaca become soiled. Dyspnœa soon sets in, and peculiar sobbing and wheezing sounds and rhonchi are frequently heard. The comb gradually turns blue. The bird becomes more and more debilitated, staggers, falls down, strives in vain to get up, and dies either comatose or convulsed.

In exceptional cases, and apparently when the infection has been present for a considerable time in a poultry yard, the

disease may assume a comparatively slow course, and may then last a week or more. During feeding experiments, Semmer and others have seen death postponed for 3 weeks. Wertheim states that he has experimentally produced in pigeons, by the inoculation of somewhat old cultures, a chronic form of typhoid from which the birds took 10 days or longer to die, with symptoms of pyæmia; although fresh cultivations caused death from septicæmia in from 12 to 24 hours.

**Anatomy.**—The principal changes are found in the intestines, heart, and lungs.

1. The external surface of the *intestinal canal* generally appears highly congested, and its mucous membrane, especially in the small intestine, is of a dark-red colour, and is covered with hæmorrhagic spots. The contents of the intestines may be watery, frothy, muco-purulent and yellow, or bloody and resembling chocolate. We also find defects in the epithelium, especially on the top of the intestinal villi, and sometimes even a croupy exudate on the mucous membrane, and the follicles of the intestines are often ulcerated. The mucous membrane of the colon, rectum, crop, and pharynx, is often greatly congested. The lymph glands of the peripheral group which is at the opening of the cæcum, is often swollen to the size of a bean, and takes the shape of the top of a mushroom.

2. The *heart*, as a rule, is covered with red points (sub-epicardial hæmorrhages), as if sprinkled over with dark-red dots, but sometimes the only change present is severe injection of the blood vessels. The pericardium usually contains a slight quantity of serous fluid, and myocarditic and pericarditic changes may also be observed.

3. The *lungs* are often found to be very rich in blood, thickened, of a dark brownish-red colour, and so heavy that they will sink in water (croupy and hæmorrhagic pneumonia). Fibrinous pleuritis and peritonitis have also been observed in a few cases. Catarrhal laryngitis, tracheitis, bronchitis, and œdema of the lungs are not rare. The respiratory changes chiefly occur in land birds; and intestinal and cardiac affections in water-fowl.

The other changes are in no way characteristic. We should remember that the flesh of birds which have died of typhoid is frequently normal in appearance, especially when the course of the disease has been very rapid. In other cases it is of a dark dun (grey-red) colour, and suffers from fatty and lardaceous degeneration. The liver sometimes shows dots. Dark-blue *post*

*mortem* spots often appear very quickly on the skin. The bacteria of typhoid are to be found in large numbers in the blood, and in all the organs of the body. In chronic cases, caseous deposits are sometimes found in the lungs and intestines as in contagious pneumonia (p. 85), in the deposits of which, the ovoid bacteria can be demonstrated (Sticker).

**Diagnosis.**—The diagnosis of fowl typhoid during life depends on the epizootic character of the disease; its exceedingly acute course; and frequency of diarrhoea. Ample data for the recognition of the disease after death are furnished by the morbid changes in the intestines, heart, and lungs, and by the presence of the bacteria. It is hardly possible to confuse the disease with diphtheritic inflammation of the mucous membrane, which has a much slower course, and which is characterised by the presence of a diphtheritic coating of the mucous membrane of the mouth. It is often more difficult to distinguish typhoid from acute poisoning, for which it is frequently mistaken by owners of birds. Such cases can be decided only by a *post mortem* examination.

**Diagnostic Inoculation.**—Kitt points out that the local anatomical changes which are observed in inoculated animals may be utilised for the diagnosis of fowl cholera. The constant characteristic change on the site of the inoculation in pigeons (which die in from 12 to 48 hours in consequence of the inoculation) consists in a yellow nodular prominence. After removing the skin, the surface of the muscles is found to be covered with patches of a dry, straw-coloured exudate, varying in size from a sixpenny piece to a shilling; and a transverse section of the muscles through the patches of exudate shows yellow discoloration and nodular induration. The site of the inoculation in fowl manifests white, lardaceous discoloration, and swelling and induration of the muscles. The bacteria are found in large numbers by themselves in the blood contained in the heart of inoculated pigeons.

**Prognosis.**—The prognosis, contrary to what is the case in fowl diphtheritis, is very unfavourable in fowl cholera, in which the mortality averages from 90 to 95 per cent. In some cases, the disease runs a milder course; probably because the contagium varies in virulence, and a certain amount of immunity is acquired by the bird having passed through a previous attack. Also, experience teaches us that early treatment considerably decreases the mortality.

**Therapeutics.**—The course of the disease is so rapid that we are often too late in administering medicines, which, apart

om this, are of less importance than prophylaxis. It is always advisable to at once administer internally a  $\frac{1}{2}$  to 1 per cent. solution of sulphate of iron or of hydrochloric acid, or a weak solution of tannin ( $\frac{1}{2}$  to 2 per cent.). We prescribe, for instance : tri sulphas, 5 parts ; aquæ destillatæ, 500 parts ; or, acidi hydrochlorici, 3 parts ; aquæ destillatæ (foeniculi *vel* menthæ), 500 parts ; and we give a tablespoonful of either of these solutions every hour to geese and domestic fowl, and a teaspoonful to pigeons. These medicines may be administered prophylactically to healthy birds in an infected poultry yard. Nocard states that he has often cut short the disease by subcutaneous injections of a 5 per cent. solution of carbolic acid.

*Prophylaxis* consists in the separation of the healthy birds from the infected (not *vice versâ*), and in a thorough disinfection of the yard or coop, which may be carried out as follows : the floor, walls, utensils, perches, etc., are thoroughly cleansed either with a hot solution of caustic potash, or, still better, with a 1 to 1,000 solution of corrosive sublimate, or a 3 per cent. solution of carbolic acid or creolin. The walls are white-washed, and the dead bodies and all fæcal matters are burned or buried at a sufficient depth. It is advisable to repeat several times this method of disinfection.

### **Protective Inoculations against Fowl Typhoid.—**

Pasteur recommended, as the most effective means of combating fowl typhoid, the inoculation of healthy birds with the bacteria of typhoid attenuated by cultivations. He found that the inoculation of attenuated vaccine produced, on the site of the inoculation, only a local swelling, which caused the muscles lying under it (the pectoral muscles) to become necrotic without suppuration. Although the inoculated birds became ill, they survived, and acquired immunity. Pasteur found that some domestic fowl required to be inoculated 2 or 3 times with the attenuated virus before they became immune. He therefore introduced a two-fold inoculation, first with a very weak vaccine (*premier vaccin*), and later on with a stronger vaccine (*euxieme vaccin*). The bacteria were attenuated by keeping the cultivations exposed to air for from 3 to 10 months, in which case, according to Pasteur's opinion, they became weakened by the oxygen contained in the air.

Cagny employed this method in 1885 for the protective inoculation of 63 healthy birds in an infected poultry yard, by first using the weak vaccine ; and 12 days afterwards, the

stronger vaccine. After the first inoculation, all the birds, with the exception of the ducks, became ill, and 9 died; and after the second inoculation, 8 died; consequently there was a mortality of 17 in 63. In a second experiment with 36 fowl, 8 died after the first inoculation, and 2 after the second; making a loss of 10 out of a total of 36. Cagny attributes the very unfavourable results of this experiment to the fact that the birds had taken the disease in the ordinary manner from the already infected poultry yard, previous to inoculation.

Kitt declares, from the experiments he made with Pasteur's protective inoculation, that it is not only useless, but is actually dangerous; because the disease is spread by it.

**Cholera of Canaries and Ducks.**—Cornil and Toupet state that canaries and ducks suffer from peculiar infective diseases which are different from ordinary fowl cholera, although they resemble it in symptoms and anatomical changes; for death ensues in 2 or 3 days with diarrhoeal muscular tremors, weakness, and paralysis. These diseases are characterised by the fact that the inoculation with the ovoid bacteria which are present in the blood, produces a fatal disease only in canaries and ducks, but not in other birds.

**Klein's Fowl Disease.**—Klein describes an avian epizootic (grouse disease), which is independent of fowl cholera, and which is caused by a bacillus with rounded ends (*bacillus gallinarum*). It commences with diarrhoea and ends in death in 30 hours. Pigeons proved immune to the inoculations. Domestic fowl died in 3 days after a period of incubation of 6 days. On *post mortem* examination the liver and spleen were found to be enlarged and softened. The very abundant intestinal mucus is almost a pure cultivation of the bacilli.

**Epizootic Dysentery.**—Lucet described under this name an infective disease that is said to chiefly attack domestic fowls and turkeys, and is manifested by depression, debility, complete loss of appetite, violent thirst, and profuse diarrhoea, which is at first mucilaginous, then bluish-grey, later on yellow, and finally sanguineous. The temperature falls 2° or 3° C. Most cases end in death, which takes place generally between the ninth and thirteenth day. This disease is supposed to be caused by a facultative bacillus, which can be easily cultivated on peptonised veal broth. It is distinguished from chicken cholera by its slower course, its slighter infectiousness, and the impossibility of transmitting it by inoculation to rabbits. It has a period of incubation of 3 or 4 days.

**Maladie du Sommeil.**—Nocard gave this name to a disease of fowl which is caused by a very small bacillus. The bacilli are particularly numerous in the spleen. The disease was at first considered to be a chronic form of typhoid. It is characterised chiefly by stupor and almost uninterrupted somnolence. The infected animals die in from 8 to 14 days.

**Vibrio-cholera of Fowl.**—This disease, which has been described by Gamaleia, is caused by a very motile, curved bacterium that has slender flagella (*vibrio Metschnikoff*). It closely resembles the cholera bacillus of man, on which account the name of "vibrio-cholera" or "gastro-enteritis cholericæ avicuum" has been given to it.

**Mycosis of Parrots.**—Wolff described under this name an infective disease of parrots which has raged for the past 15 or 20 years among recently imported grey parrots (*psittacus erithacus*, and *jako*), and which kills them by thousands. The infection is said to take place on board ship during the voyage from the west coast of Africa. It is caused by micrococci, namely, by the *streptococcus pernicius* (Zopf). Its development, according to Wolff, is favoured by damaged food, bad drinking water, dirty and unsanitary condition of the cages, and bad air in the holds of the ships. The symptoms of this disease, which generally ends in death, are: decreased appetite, debility, depression, drooping of the wings, diarrhœa, in rare cases vomiting, squatting on the floor, and finally convulsions. On *post mortem* examination we find in the liver, and less frequently in the lungs, spleen, and kidneys, characteristic grey and dirty-white hard nodules which are about the size of millet seeds, and which contain the pathogenic micrococci in great numbers. Local necrosis is caused by the cocci settling in the tissues. Also, slight enteritis and sometimes lobular-pneumonic changes are met with.

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#### DEER AND CATTLE DISEASE (*Septicæmia Hæmorrhagica*, and *Buffalo Disease*).

[Dr. Lingard (*Annual Report of the Imperial Bacteriologist* for 1902-1903) gives the following synonyms for this disease: *Barbone dei bufali* (Italy), *Wild und Rinderseuche* (Germany), *Pasteurellosis bovis* (France), Malignant sore throat (India), *Ghotwa*, *Gharwa*, or *Galghotu* (Southern Punjab), *Khoumnaq* (strangles) Egypt, *Sakit Ngorok* (Exanthemous) and *Koerang Naplas*, (pectoral form) Java. He tells us that of all animals experimented with, rabbits are the most susceptible to the organism of hæmorrhagic septicæmia.

"Animals which proved refractory to inoculated virulent cultures of hæmorrhagic septicæmia, viz., ponies, sheep, goats, and protected bovines and buffaloes, when subsequently injected for a long period with successively increasing doses of such cultures, furnished powerful protective sera against hæmorrhagic septicæmia" (Lingard).—Tr.]

BOLLINGER described under this name (*Wild und Rinderseuche*) an epizootic which broke out during 1878 in the neighbourhood of Munich, and first occurred enzootically among the deer of several royal parks, and later on also epizootically, especially

in cattle. The disease, which was at first confined to the deer and wild pigs that were kept in parks, soon spread to the cattle that were in the neighbourhood, and in some cases also to horses and domestic pigs. Altogether, 387 head of game died in the parks, namely, 153 red and fallow deer, and 234 wild pigs. The epizootic recurred in the following summers, and spread widely among the domestic animals of Upper Bavaria, especially during 1881. In subsequent years, it was restricted to certain localities. In 1889 it raged throughout the district of Bayreuth. It appears to have occurred among cattle in other countries, as for instance, in the Prussian district of Schlüchtern, where formerly there was an average yearly loss of 100 from it; but only of 11 in the year 1885-1886. According to Jakobi, 34 out of 40 cattle which were grazing in the forests of Obornik died from it (principally from the exanthematic form) during the spring of 1889. During 1894, in the park of the Prince of Sigmaringen, 148 fallow deer and 25 other deer died of it (Hoffmann).

This epizootic was evidently well known in former times, as far as its complex symptoms were concerned. Thus, the "terrible" epizootic among cattle and red deer which was described by "The Veterinarian" in 1854, was certainly this deer and cattle disease. It was usually mistaken for anthrax, with the lingual form of which it possesses in some cases great similarity. The question whether the majority of the cases described as "anthrax of the tongue" or "gloss-anthrax" were not those of this disease, is almost self-suggestive.

**Etiology and Pathogenesis.**—We have no exact information about the contagium.\* We may here refer to the remarks made further on, about the oval bacterium of septicæmia hæmorrhagica (*bacterium multacidum*), which was discovered by Kitt. Friedberger and Hahn were the first to transmit by inoculation the disease from infected cattle to horses, which died from it, and also to cattle, which manifested only a local swelling. Friedberger and Frank found micrococci and small bacilli in the serum of the œdema. Bollinger successfully inoculated the disease in horses, goats, pigs, sheep, and rabbits. He pro-

\* "The organism, the cause of the disease, is a short bacillus, ovoidal, with rounded extremities. The organism when stained presents the two terminal portions coloured with the intervening or middle portion uncoloured. The bacillus varies considerably in size, the largest specimens having been always obtained from the spleen of the rabbit" (*Lingard*).

duced in cattle the pectoral form by feeding with the sanious contents of the intestines of animals which had died in 54 hours from the exanthematic form, and produced the exanthematic form by inoculation of the blood and pleuritic exudate of animals which had died of the pectoral form ; thus proving the identity of these two forms of the disease. No positive case of its transmission to man has been observed, and no bad results have been caused by wounds received during *post mortem* examination, or by the consumption of the flesh of infected animals.

The exciting cause of the disease seems to be able to enter the body by the skin (exanthematic form), lungs (pectoral form), and intestines (intestinal form). Bollinger's inoculation experiments prove that the pectoral form may occur after the absorption of the contagium from the intestines. Wild pigs possibly become infected by eating the bodies of animals which have died from this disease. The exanthematic form appears to be caused chiefly by the penetration of the contagium into small wounds of the skin (stings of flies and hornets, injuries on the head and in the oral cavity). A direct transmission from one animal to another has never been known. The disease has also been disseminated by the sale of the meat and skins in other villages. Zeiliger reported even a case of a man who had been employed to search for the carcasses of the deer that had died from this disease, introducing it into his own cow-shed.

**Anatomy.**—From the anatomical changes in the principal organs, we can distinguish three forms of “deer and cattle disease,” namely, exanthematic, pectoral, and intestinal. The first two are the most important, and are frequently complicated with the third. In some cases, all three forms are present at the same time.

1. The *exanthematic* form manifests itself first by a considerable swelling of the skin and subcutaneous connective tissue. The skin is oedematously swollen on the affected parts (head, intermaxillary space, and neck), and the subcutaneous connective tissue is frequently sero-gelatinously infiltrated to an enormous extent, and thickened. In the meshes of the subcutaneous connective tissue, we sometimes find a clear, amber, or gold-coloured serum ; at other times it is infiltrated with hæmorrhages of greater or less size. The lymph glands (submaxillary and upper cervical glands) in the neighbourhood of these swellings are partly serously, partly hæmorrhagically infiltrated,



and are greatly enlarged. The mucous membrane and the submucosa of the oral cavity may show similar changes, in which case, they become cyanotic and considerably thickened. In particular, the mucous membrane on the sides of the tongue often forms gelatinous, loose pads. The tongue itself is sometimes enlarged out of shape. On one occasion, Friedberger found the tongue of an ox to be 4 inches thick, and to weigh nearly 9 pounds. Its surface varied in colour from purple to a dirty brown-red, and under the epithelium were found dark red effusions which were the size of the palm of a man's hand, and which extended into the muscles of the tongue. The tissues of the tongue contained a large amount of juice, and a bright yellow fluid which resembled plasma and which rapidly coagulated, flowed from the purple-coloured section. The mucous membrane of the pharynx frequently shows similar changes. In these cases, the salivary glands appear to be very anæmic and dry. The respiratory mucous membrane may be swollen in the same way. Thus we find in the trachea, bronchi, and especially in the larynx, loose projections of the mucous membrane, which considerably narrow the lumen of these passages. Croupy changes have also been seen in the bronchi. Hæmorrhages are found in various organs, especially under the serous membranes, in the muscles, and in the lungs. The spleen is perfectly normal, except that its pulp is somewhat drier than usual. The blood is almost always of a normal colour and consistency. The perirenal connective tissue is frequently infiltrated with blood. Lastly, we almost always find symptoms of hæmorrhagic enteritis.

2. In the *pectoral* form, the interstitial tissue of the lungs is gelatinously infiltrated, and the lungs themselves are in a condition of hepatisation (croupy pneumonia). The pleuræ are inflamed, swollen and covered with sero-fibrinous and purely plastic deposits (*pleuritis serosa et sero-fibrinosa*). A fluid exudate of varying quantity (in one case 30 litres) is found in the thoracic cavity. Pericarditis and mediastinitis are also present. Also in this form, we find hæmorrhages everywhere in the body, and frequently hæmorrhagic enteritis. The spleen and the blood are normal.

3. In the *intestinal* form, the mucous membranes of the intestines, especially of the small intestine, and sometimes that of the stomach, are swollen; the epithelium is desquamated; the mucous membrane is infiltrated with hæmorrhages of various extent (enteritis and gastritis hæmorrhagica); and a thin and

sanious fluid is contained in the intestines. These changes are usually complicated by appearances of the exanthematic or pectoral form. *Post mortem*, we find only extremely slight changes in very acute cases.

**Symptoms.**—Two forms, the exanthematic and the pectoral, can be distinguished clinically.

1. The *exanthematic form* is the one which usually attacks cattle, and generally begins with loss of appetite and diminution in the secretion of milk. The internal temperature is very high, and varies on an average from  $40^{\circ}$  to  $42^{\circ}$  C. The soft parts of the head, especially of the face, intermaxillary space, dewlap, and neck, are swollen, often to an enormous size. The swelling is tense, hard as a board, hot, painful, and rarely yields to the pressure of the fingers. As a rule, it disfigures the above-mentioned parts in a very striking manner, and may attain a thickness of 2 decimetres. Symptoms of stomatitis and pharyngitis appear at the same time. Salivation is set up; a glassy mucous discharge flows in strings from the mouth; and the patient has difficulty in swallowing, and sometimes performs continued masticatory movements. Frequently the tongue hangs out of the mouth, is swollen to twice or thrice its ordinary size, is of a purple colour, and sometimes shows the impressions which the teeth have made on it. The mucous membrane on the sides of the tongue sometimes forms loose, gelatinous projections. Difficulty of breathing, and even actual suffocation, may occur in consequence of the great swelling of the oral and pharyngeal cavities. The other visible mucous membranes of the head are of a brown-red colour and infiltrated with hæmorrhages; and the vaginal mucous membrane may be greatly congested. Towards the end of the attack, excessive dyspnœa and colic set in. The patient strains to defecate, groans, frequently lies down, and may even pass croupy casts of the intestine along with the fæces, which, later on, take the form of severe diarrhœa. There is increasing weakness, the animal persistently lies down, and death takes place in from 12 to 36 hours; the minimum duration of the disease being 6 hours, and the maximum, 3 or 4 days.

2. The *pectoral form* is the usual one which occurs in wild deer. Exact observations of the disease are naturally wanting. The symptoms which were observed in some cases in cattle were, on the whole, those of a rapidly-running inflammation of the lungs with dyspnœa. Its duration is longer than that of

the exanthematic form, and amounts on an average to 5 or 6 days, and may even extend to 8 days.

**Prognosis.**—The prognosis in cattle (no prognosis is possible with wild deer) is very unfavourable. According to Bollinger, the mortality is decidedly higher than that of anthrax. Putscher saw only 9 recoveries among a total of 95 head of cattle; and Jakobi, only 6 among 40.

**Differential Diagnosis.**—"Deer and cattle disease" has been confused chiefly with anthrax and pleuro-pneumonia contagiosa.

1. The similarity to *anthrax* consists in the carbuncular swellings of the skin and oral mucous membrane; development of hæmorrhagic enteritis; and occurrence of hæmorrhages in all the organs of the body. Not very rarely, anthrax also occurs epizootically among deer kept in parks. Bollinger, for instance, calls attention to the epizootic of anthrax which raged in 1874 in Grunewald, near Berlin, and caused the death of 2,000 head of deer. In spite of this similarity, "deer and cattle disease" can be easily and with certainty distinguished from anthrax by the following facts: absence of the bacillus of anthrax; absence of enlargement of the spleen and of the characteristic tarry condition of the blood, which are always present in anthrax; easy transmission to pigs, which are rather refractory to anthrax; difficulty of transmitting it to sheep, which can be inoculated with anthrax with the greatest ease; innocuousness of the flesh when consumed by man; and immunity of man to "deer and cattle disease."

2. Although "deer and cattle disease" is often confused with *pleuro-pneumonia contagiosa*, especially at the beginning of an outbreak, such a mistake may be avoided without fail by paying attention to the anatomical conditions. The lung changes in "deer and cattle disease" are of a perfectly uniform character, and are of the same age; thus differing from the irregular lung changes in pleuro-pneumonia, the course of which is much slower. According to Kitt, the period of incubation of "deer and cattle disease" is far shorter, and the rapidity of its dissemination is much greater. In pleuro-pneumonia we find only pectoral symptoms, which are frequently entirely absent in cases of "deer and cattle disease." Also, calves can suffer from "deer and cattle disease," which differs from pleuro-pneumonia by the easy and typical manner it can be trans-

mitted. Very serious and far-reaching consequences may ensue, as regards inoculation, by mistaking the pectoral form of "deer and cattle disease" for pleuro-pneumonia.

3. Mistakes may also arise with *malignant œdema*, which, however, can be transmitted only by subcutaneous inoculation; though "deer and cattle disease" can be conveyed by feeding and by cutaneous inoculation. The same remark applies to *quarter-ill*, which is sufficiently distinguished by its stationary and narrowly limited occurrence, and the crackling character of its tumours. There is no difficulty in distinguishing "deer and cattle disease" from *rinderpest*.

**Therapeutics.**—Up to the present, the treatment of cattle suffering from "deer and cattle disease" has been entirely unsuccessful. Friedberger tried in one case a subcutaneous injection of a 10 per cent. solution of carbolic acid, and the internal administration of salicylic acid, but with negative results. It might be well, as in *quarter-ill*, to make free incisions into the swellings, with subsequent application of strong disinfectants. The German veterinary police regulations for "deer and cattle disease" are the same as those for anthrax.

**Septicæmia hæmorrhagica.**—In 1885, Kitt found in the blood and in the tissues of cattle, horses, and pigs which had died in the district of Sembach of an unknown epizootic, non-motile, oval bacteria of  $0.6\ \mu$  in length and  $0.3\ \mu$  in breadth. These bacteria could be stained only at their ends, and were present chiefly in the blood-serum. After inoculation, they proved to be pathogenic to mice, rabbits, pigeons, small birds, pigs, goats, horses, dogs, sheep, and cattle; and in all cases set up a hæmorrhagic tracheitis in rabbits. They were similar to the bacteria of septicæmia of rabbits, chicken cholera, and contagious pneumonia of the pig. Kitt stated that this disease is identical with "deer and cattle disease," because the two diseases show similar pathological and anatomical results, and can be transmitted to the same animals. He also found that the bacteria in question closely resembled those that were present in samples of blood which had been taken during the epizootic of 1878 from animals that had died of "deer and cattle disease." Hueppe, from the examination of the material which had been sent to him by Kitt, confirmed Kitt's observation with respect to the above-mentioned unknown infective disease, and obtained similar results by inoculating the bacteria in small animals. He also considers that these bacteria are identical with those of "deer and cattle disease," which, according to him, may occur as a pure septicæmia, intestinal mycosis, or infective pleuro-pneumonia. He suggests for it the name of "septicæmia hæmorrhagica." Hueppe also inoculated rabbits, guinea-pigs, and mice, with the respective cultivations of the bacteria of swine fever, the infective disease investigated by Kitt, chicken cholera, and septicæmia

of rabbits; and states that he found absolutely identical changes, and similar behaviour of the respective bacteria in the fluids of the tissues and in the blood, in all these diseases. Consequently he considers contagious pneumonia of the pig, chicken cholera, septicæmia of rabbits, "deer and cattle disease," and contagious equine pleuro-pneumonia to be merely different forms of his "septicæmia hæmorrhagica."

Jensen described a disease of calves which resembled "deer and cattle disease" in cattle, and in which the same oval bacteria were found. In a herd of cattle in Jutland, 16 calves about 2 months old suddenly died after having been 12 to 24 hours ill, with symptoms of high fever ( $41^{\circ}$  C.) and diarrhœa. *Post mortem* examination showed gastro-enteritis, fibrinous pleuritis, pericarditis and numerous ecchymoses under the epicardium. Inoculation experiments produced acutely fatal septicæmia in calves, rabbits, and mice. All the inoculated rabbits became affected with hæmorrhagic tracheitis and considerable swelling of the spleen. Bongartz reports a similar disease of calves. Buch observed 3 cases of sporadic hæmorrhagic septicæmia in full-grown cattle with similar bacteriological results. Guillebeau and Hess met with in cattle a very grave form of hæmorrhagic septicæmia which assumed the aspect of acute pneumo-pleurisy, with, in some cases, swellings on the head. The bacillus, however, was not identical with that of bovine "deer and cattle disease."

**Buffalo Disease.**—An epizootic disease of buffaloes is known in Italy under the name of "barbone." It is chiefly found in the districts of Salerno, Rome, and Terra di Lavoro, and closely resembles "deer and cattle disease."\* It was first described from a veterinary standpoint by Metaxa in 1816, and was regarded, later on, as typhus, anthrax, or strangles. We are indebted to the more recent investigations of Oreste and Armanni for exact information respecting its pathology. Barbone occurs chiefly in summer among buffalo calves. It begins with high fever ( $41^{\circ}$  to  $42^{\circ}$  C.), stupor, loss of appetite, characteristic swelling of the intermaxillary space, salivation, mucous discharge from the nose, swelling of the mucous membrane of the mouth and tongue, and excessive dyspnoea. The swellings, which may also appear on the abdomen, throat, face, and limbs, pit on pressure with the fingers, and do not crackle. If we make an incision into them, we find that they are produced by a yellow gelatinous exudation. In very acute cases, the animals die in 3 to 6 hours. Usually they fall down in from 10 to 24 hours, and die in convulsions. Recovery may take place, if the animal remains alive over 24 hours. The duration of the disease in a district is generally 9 or 10 days, and the mortality is 40 or 50 per cent. On *post mortem* examination, the subcutaneous connective tissue of the swollen parts and the interstitial connective tissue of the muscles lying under them, are found to be infiltrated with a yellow gelatinous exudate. The spleen is unaffected. We find in the blood, ovoid bacteria, which very closely resembles the bacteria of swine fever, fowl cholera, and septicæmia of rabbits. The disease can be transmitted to horses, cattle, sheep, pigs, guinea-pigs, rabbits, pigeons, and turkeys, but not to dogs. The virus is found not only in the blood

\* Linguard describes them as the same disease.—TR.

and exuded matter, but also in the saliva, urine, milk, and foetal blood. Natural infection takes place by small injuries of the skin, alimentary canal, and less frequently by the lungs (compare its similarity to "deer and cattle disease"). The contagium is destroyed by drying, boiling water, 2 per cent. solution of carbolic acid, and 5 per cent. solution of sulphuric acid. Oreste and Armanni recommend inoculation as a prophylactic measure. They succeeded in attenuating the virus by the inoculation of pigeons, and produced immunity in buffaloes by inoculating them, on three occasions, with very minute quantities of the blood of inoculated pigeons. They state that during the years 1888-91, inoculation was performed on 1,986 buffaloes, out of which only 6 per cent. died; although formerly from 45 to 50 per cent. succumbed to the disease taken in the ordinary manner. It seems from this that protective inoculation has excellent results.

Buffalo disease has been proved to have occurred recently in Hungary. During 1891, 4 per cent. of all the buffalo calves suffered from it in Siebenbürgen. In 1892 the disease attacked 237 animals, out of which 231 died (Hutyra). Reischig regarded this malady as identical with bovine "deer and cattle disease." Makoldy and Sequens, on the contrary, consider that it is swine fever; because, in the same farm-yards, buffaloes and pigs simultaneously exhibited symptoms of swine fever, while cattle remained unaffected. Gál recommends injections of 5 per cent. solution of creolin, and the internal administration of creolin in doses up to 50 grammes. Under this treatment, 5 buffaloes out of 17 recovered.

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#### QUARTER-ILL.

Quarter-ill in cattle—Quarter-ill in horses.

**History.**—Quarter-ill (symptomatic anthrax, or black-leg) was described in former years, under various names, as a form of anthrax, and in France is called *charbon symptomatique*. It has long been known that this disease is not infectious, and that the meat is quite innocuous to man and beast. Even in former days, very exact clinical descriptions of the disease were made; as, for instance, that by Wallraff in 1856, which, as regards symptoms, is in no way inferior to our present knowledge of the phenomena of this disease. Also, Pfisterer, in 1870, came to the conclusion that the nature of quarter-ill is different from that of anthrax. Feser and Bollinger have, however, been the first to teach with certainty, by their etiological researches, that quarter-ill and anthrax are two entirely different diseases. Feser fully investigated quarter-ill while studying anthrax in the Upper Bavarian Alps in 1875. He became acquainted with quarter-ill in 1860, and states that even at that time he found

slender motile rods in the muscle juice. He points to small, thin, motile, serpentine rods which have rounded ends, as the cause of the disease. By the subcutaneous inoculation of the mud of certain places in the Alps in which the disease had been very common, he produced its characteristic symptoms in cattle, sheep, and rabbits. In the same year Bollinger discovered the bacillus of quarter-ill in the form of a thin, long rod with rotatory movements; and produced the disease experimentally in cattle, sheep, and goats by the subcutaneous inoculation of the blood of animals suffering from quarter-ill.

We are indebted to Arloing, Cornevin, and Thomas for the recent investigations on the nature of the virus of quarter-ill. They have fully described its bacillus, and have clearly demonstrated its biological relations. The introduction of protective inoculation against quarter-ill was the most important result of the inoculation experiments made by them. The very recent labours of Kitt and Kitasato on the bacteriology of quarter-ill are worthy of mention.

**Occurrence.**—Quarter-ill is a stationary infective bovine disease, that is to say, it is confined to certain localities and sheds, where it remains throughout the year. It is specially active during the warmer months (June, July, and August), and occurs chiefly on swampy ground. It also affects sheep, goats, and very rarely, horses. It occurs in various places in Germany, Austria, Bavaria, Switzerland, Hungary, France, Belgium, Holland, Northern Italy, Russia, Norway, Algiers, South Africa, Australia, and other countries.

[Pease states that quarter-ill causes great loss during the rainy season in the swampy districts of the Punjab and North-West Provinces of India.—TR.]

As a rule, only cattle between the ages of 3 months and 4 years become affected. Calves under 3 months, and indigenous cattle over 4 years are rarely attacked. The reason of this, perhaps, is that the former are not exposed to the infection on account of their food being restricted to milk; and that the latter are mostly immune from having had the disease (Arloing). Moreover, calves are also much less susceptible to the artificial inoculation of the disease than full-grown animals. For instance, they can bear with impunity an amount of inoculation material which would certainly kill mature cattle. If

foreign animals are put on the infected pastures, they may become infected at any age.

One attack confers absolute immunity. It seems that pigs, dogs, cats, rabbits, grey rats, mice (at least to a certain extent), and man are naturally immune to the disease. This statement agrees with the well-known fact that the flesh of cattle which had suffered from quarter-ill can be consumed by man, dogs, and pigs without any ill result. Animals which are immune to quarter-ill are also immune to malignant œdema. The inoculation of horses, asses, and white rats, produces only a local reaction in the form of a swelling. On the other hand, quarter-ill can be easily transmitted by inoculation to cattle, goats, sheep, and guinea-pigs. In some localities, for instance, in the Upper Bavarian Alps, quarter-ill occasionally occurs simultaneously with anthrax. Hence it was looked upon in former times as a precursor of anthrax.

**Statistics.**—In Germany 1,772 cattle and 3 horses became infected with quarter-ill during the years 1891 to 1893; in Baden, 375 cattle from 1886 to 1890; in Austria, 376 cattle in 1886; and in Switzerland, 533 in 1893. Schmitt reports that quarter-ill caused very severe losses in the district of Mörs (Rhenish Prussia), especially during the early eighties after the great inundations, so that, during these years, 12 or 13 per cent. of all the young cattle died from this disease. In the Canton of Berne, 1,728 cattle died from it during the years 1882 to 1884, thus showing a yearly loss of £5,000 during that time. In the Transvaal, quarter-ill, which is locally known as *spons ziekte* (sponge disease), *okapiranka*, or *ongamero*, is said to occur throughout the whole year, and to kill 50 per cent. of all the young cattle (Theiler and Sander).

**Bacteriology.**—The bacilli of quarter-ill (*bacilli sarcophysematos bovis*) are very small, being only from 3 to 6  $\mu$  in length and 1  $\mu$  in thickness, and are therefore about the length of the diameter of a red blood-corpuscle. They show lively, rotatory, or serpentine movements, and contain, usually at one end, a bright oval spore, the greater thickness of which makes the rod assume the shape of a club. Contrary to the behaviour of other bacilli, they can be stained with Gram's solution. They are found most abundantly in the affected subcutis and muscles, but seldom in the blood; hence inoculations with blood are generally negative. They occur in great quantities in the bile and in the contents of the intestines (Kitt). In the blood, they are only found when the dead body has been kept for a considerable time. Consequently, the bacillus of quarter-ill, like that of malignant œdema, is an anærobe, which forms gas while growing in the



tissues; it does not require for its development the oxygen which is in the blood; and it forms spores within the dead body (endogenous development). It is probable that it also develops ectogenously in the soil, although this supposition has not yet been positively proved. Arloing was the first to obtain a pure culture of the bacillus, which he did in chicken broth. Pure cultures are characterised by the abundant formation of gas; thus producing a large quantity of pearly bubbles and froth, which clear away in from 1 to 3 days, and leave a thick white sediment. The bacilli of quarter-ill being anærobes, cannot be cultivated on potatoes in the ordinary way. The cultivations on gelatine are, however, very typical; as they show arborescent ramified offshoots with clefts and cavities. Ehlers has described several forms of development of these bacilli. Kitasato states that they grow best at a temperature of from 36° to 38° C., but are able to grow even at a temperature between 16° and 18° C. The higher the temperature, the quicker spore-formation takes place. In the animal body, spores do not become developed until from 24 to 48 hours after death.

The investigations of Arloing, Cornevin, and Thomas have shown that the virus of quarter-ill is exceedingly resistant to external influences, especially when it has been dried, in which condition it will continue effective for a couple of years. Hence the dried flesh remains infectious for a very long time. Burying the dead body does not destroy the contagium even in 6 months. The bacilli of quarter-ill is in no way influenced by the bacteria of putrefaction, by those of anthrax, or by severe cold. The dried and finely-powdered flesh of animals which have suffered from quarter-ill can resist steam even at 100° C., which degree of heat only weakens the virus, but does not destroy it. On the other hand, the fresh virus is destroyed by a temperature of 100° C. in 20 minutes, and by boiling water in 2 minutes; but the dried virus is destroyed only by a temperature of 110° C. in 6 hours, and by boiling water in 2 hours. The dry virus is also destroyed by the vapour of bromine; watery solution of corrosive sublimate (1 to 5,000), salicylic acid (1 to 1,000), thymol (1 to 800), carbolic acid (1 to 50), boric acid (1 to 5), hydrochloric acid (1 to 2), etc. Disinfectants, such as an alcoholic solution of carbolic acid, quicklime, sulphate of iron, chloride of zinc, and sulphuric acid, which are useful in other diseases, have proved to be ineffective. It has been found that certain chemicals, and, as already stated,

a continued high temperature weakens the virus. The virulence of such attenuated bacilli is increased by the addition of lactic acid, and, according to Nocard and Roux, by that of lactate of potash, acetic acid, dilute alcohol, etc.

**Pathogenesis.**—Quarter-ill is a wound-infection disease, that is to say, an infective disease due to the absorption of the bacilli of quarter-ill into the body by injuries of the skin or mucous membrane. These injuries, in order to afford the bacilli entrance into the body, must, as in the case of the bacilli of malignant œdema, penetrate into the subcutaneous connective tissue or submucosa ; for mere penetration of the skin or mucous membrane is insufficient. It is supposed that such injuries are inflicted, especially on the legs and mouth, while the animal is grazing, and that they come in contact with the contagium, which is present in the soil. Hess found injuries of the skin on the pasterns and cannon bones in animals suffering from quarter-ill, and is of opinion that infection of the injured oral mucous membrane takes place when grazing, chiefly while the milk-teeth are being shed. Hafner states that the oral and pharyngeal cavities form a frequent gate of entrance for the bacilli, particularly in stall-fed animals. According to the inoculation experiments of Arloing, Cornevin, and Thomas, an infection from the cow to the fœtus can take place. The calves of cows which have had quarter-ill seem to be immune.

Inoculation experiments have shown that the period of incubation averages 2 days, with a minimum of 1 day, and a maximum of from 3 to 5 days.

**Symptoms.**—Quarter-ill has a very rapid course, which, with few exceptions, ends fatally in from one and a half to three days. It is characterised by a rapidly increasing swelling of the skin, which crackles on being touched ; high temperature ; secondary swelling of the lymph glands of the skin ; and secondary motor-disturbances.

1. *The swellings of quarter-ill* may appear on different parts of the body, and are chiefly found on the upper parts of the thighs, neck, shoulders, lower region of the chest, loins, and sacrum. They never appear below the hocks and knees. They are less frequently found on the palate, at the base of the tongue, and in the pharyngeal cavity. The swelling is at first very small and painful. It rapidly spreads, may attain extraordinary size in a few hours, and may even extend over the whole trunk.

These swellings are then characterised by a crackling and gurgling sound when the hand is passed over them or when they are kneaded with the fingers. They give on percussion a clear, tympanitic sound, and in their centre are insensitive, very dark-coloured, dried up like parchment, or even gangrenous. They become strikingly cold, and no pain is felt by the animal when an incision is made into them, in which case a dark-red frothy, unpleasantly smelling fluid flows from the wound. Sometimes, only one swelling of greater or less size appears; but more frequently we find several, which not uncommonly become confluent. The lymph glands in their neighbourhood are greatly swollen and can then be distinctly felt as tumours under the skin.

2. The chief *general symptoms* are as follows: sudden suppression of appetite and rumination, debility, depression, high internal temperature (up to 42° C.), lameness, stiffness, and dragging of one leg in consequence of the swelling spreading to a limb. According as the swelling becomes larger, the other symptoms increase in intensity. The breathing becomes accelerated and difficult, and the animal groans, and is sometimes seized with violent attacks of colic. Death occurs with increased weakness and fall of the internal and external temperature of the body.

In some cases the general symptoms are the first to appear; in others, the characteristic swelling of quarter-ill takes the initiative. A few mild cases, which showed slight swelling and moderate fever, and recovered in 24 hours, have been observed in aged animals. Arloing mentions a mild form of quarter-ill which is chiefly manifested by loss of appetite, slight colic, tympany and debility.

**Anatomy.**—The *skin* covering the swellings of quarter-ill show the already described changes of dry gangrene. The subcutaneous connective tissue is yellow, gelatinous, infiltrated with blood, and contains bubbles of gas, which escape if an incision be made into the tumour. The muscles underneath the tumours are of a dirty brown or even of a black colour. At other places they are dark red or dark yellow, and when exposed for some time to the air, they may have a golden lustre. They are brittle, rotten, porous, and very rich in fluids. They crackle on palpation or when incisions are made into them, and blood of a frothy, greasy, tarry condition and of a sickly fœtid smell issues from them when they are squeezed. The fibres of the muscles show extremely varied degenerative changes. The gases that

are present in the muscles are inflammable and burn with a bluish flame on being ignited. They are stated to have but little smell, or to possess an odour which is not particularly bad ; on which account it is assumed that they consist of carburetted hydrogen. They are also said to contain carbon dioxide, but no oxygen. An exact chemical analysis of these gases has not yet been made, although it would be very desirable. As some observers state that stinking gases are given off when incisions are made into the muscles, it is not improbable that the muscles also contain sulphuretted hydrogen. The lymph glands near the tumour are enlarged, and full of blood ; they contain hæmorrhages, and are sero-saniously infiltrated. The afferent lymph vessels are sometimes distended with gases, and then have the appearance of strings of beads. We find changes, similar to those of the external muscles, in the muscles of the tongue and pharynx, when the disease, as may happen in rare cases, is localised in the oral and pharyngeal mucous membranes.

In the *abdominal cavity* we frequently find a large amount of blood-red exudate ; but in other cases, only small quantities of a serous fluid, or even no changes at all, according as the swelling of the muscles has or has not spread to the peritoneum. Yellow gelatinous and hæmorrhagic infiltrations are often met with in the omentum, mesentery, and neighbourhood of the kidneys. The mucous membrane of the stomach and small intestine is frequently swollen, congested, and infiltrated with hæmorrhages, in which case the contents of the intestine are bloody. The liver is hyperæmic, but the spleen is perfectly normal.

In the *thoracic cavity* we sometimes find the pleuræ in the neighbourhood of the swollen parts of the skin and mediastinum to be hæmorrhagically infiltrated. The pleuræ also shows large ecchymoses, in which case the thoracic cavity contains a sero-sanious exudate. Hæmorrhages are also present in the lungs, pericardium, myocardium, and under the endocardium. The muscular tissue of the heart is very soft, but the other muscles show only slight changes. The mucous membrane of the bronchi are sometimes hyperæmic and hæmorrhagically infiltrated.

The *blood* is of a normal colour and readily coagulates. The fluids of the muscles have, according to Feser, an acid reaction, and the flesh becomes rapidly putrid. The dead body is greatly inflated by the gases that accumulate under the skin. The

bacilli of quarter-ill are found only in small numbers in the blood during life, but abundantly a few hours after death.

**Differential Diagnosis.**—We have here to consider only the differentiation of quarter-ill from *anthrax*. With respect to the differential diagnosis of malignant œdema, see p. 7.

The chief diagnostic characteristics are as follows :—

1. Quarter-ill is *clinically* distinguished from anthrax by its characteristic crackling and gas-containing tumours, which never occur in anthrax ; by its non-contagious nature ; and by the greater severity of its course.

2. It is *anatomically* distinguished by the above-described changes in the muscles, which always contain gas, and by the normal condition of the blood of the spleen. It is also stated that the blood of quarter-ill, contrary to what is the case with the blood of anthrax, always coagulates completely.

3. It is *bacteriologically* distinguished by the bacilli being different. Those of quarter-ill are shorter and thicker, have rounded ends, are very motile, contain spores at one end, and are consequently club-shaped. The bacilli of anthrax, on the other hand, have square ends, are of uniform thickness, are non-motile, and have characteristic pure cultivations.

4. Cutaneous *inoculations* with the virus of quarter-ill give negative results ; but even a very small quantity of the virus of anthrax introduced into a wound of the skin is effective. Subcutaneous inoculations of quarter-ill cause on the site of the inoculation a very severe swelling, which never occurs to the same extent with inoculations of anthrax. Intravenous inoculations of quarter-ill produce only a slight affection with subsequent immunity, but similar inoculations of anthrax cause certain death. Guinea-pigs always succumb to inoculations of both anthrax and quarter-ill. Rabbits are immune to quarter-ill, but are exceedingly susceptible to anthrax. In order to be able to positively distinguish anthrax from quarter-ill, we should inoculate simultaneously a rabbit and a guinea-pig. If only the guinea-pig dies, the disease is quarter-ill ; if both die, it is anthrax. The predisposition to quarter-ill in animals is generally less than that to anthrax.

Quarter-ill may also be confused with *septic metritis*. The differentiation in this case is also bacteriological.

**Therapeutics.**—Quarter-ill is such a malignant disease, and its course is so very acute, that treatment is generally out of

the question ; although, if we take the case in hand at the beginning of the attack, we may attempt a cure by making free incisions into the swollen parts of the skin, and by introducing strong disinfectants into the subcutaneous connective tissue. Wallraff states that he has heard of animals being saved, when the disease starts in a leg, by applying, as soon as the swelling begins, a tight ligature around the limb just above the enlargement, which should then be scarified. We cannot deny the possibility of a favourable result from this treatment. Tisserand tells us that, during 1877 in France, 32 head of cattle recovered from quarter-ill out of a total of 1,875 (not quite 2 per cent.).

The chief *prophylactic means*, if practicable, is to avoid those pastures which have been known to have produced the disease. In Upper Bavaria, for instance, a pasture land which was thus infected was converted into a forest (Zeilingner). As infection usually takes place by wounds of the skin and not from the intestine, Kitt proposes to feed the cattle in sheds with the hay made from the grass of infected places, and to exclude from pasture lands cattle which have wounds on their pasterns or which are shedding their milk-teeth. It is, however, true that the disease not uncommonly occurs in cattle which are exclusively stall-fed, as in Baden and Württemberg. The best means of combating quarter-ill is the application of effective veterinary-police regulations. It may be reasonable to apply to quarter-ill almost the same regulations as those for anthrax, as has been done in several countries, for instance, in Prussia, Württemberg, and elsewhere. In Austria, the following special measures have been put into force : compulsory notification ; prohibition of slaughter ; prohibition of using the dead body, with the exception of the skin ; rendering the dead body and the offal innocuous ; disinfection ; and periodical inspection.

**Protective Inoculation of Quarter-ill according to Arloing, Cornevin and Thomas** (*Lyons method*).—The protective inoculation method introduced by Arloing, Cornevin and Thomas, is of great scientific interest and practical importance. These authorities found that, although the subcutaneous and intramuscular inoculation of the virus almost always causes death, intravenous and also tracheal inoculation produces only a harmless general affection which is followed by immunity. On the other hand, all feeding experiments which were made gave no result. Working by the light of these observations, they

inoculated 13 animals at Chaumont in 1880, by injecting, directly into the jugular vein, filtrated quarter-ill virus which had been dissolved in distilled water, with the result that the animals thus protected proved, 6 months later, to be quite immune to a subcutaneous inoculation of the virus; whilst out of 12 non-protected animals which were also subcutaneously inoculated with the virus, 9 died of quarter-ill and 2 became extremely ill. In 1881 they inoculated, with a similar good result, 245 animals in the Département de la Haute-Marne; and 78 during 1882, in the Département de l'Ain.

The intravenous method was found to demand such great attention to detail—for the jugular vein had to be laid bare on each occasion, and very exact precautions had to be taken when introducing and withdrawing the syringe, to avoid the penetration of the inoculation material into the perivascular connective tissue—that they discarded it for inoculation of the subcutis of the tip of the tail; because they found that this method produced only a temporary, harmless swelling, which was also followed by perfect immunity. They explain that the mild character of the reaction at the tip of the tail is due to the density of the connective tissue at that part, and to its low temperature, both of which facts more or less check the dissemination of the bacilli in the connective tissue spaces. Arloing, Cornevin and Thomas also attenuate the virus by the influence of heat before inoculating. The inoculation experiments made during the last few years in France, Switzerland, Austria, Germany and other countries indisputably prove that Cornevin's protective inoculation greatly reduces the mortality of quarter-ill.

**Preparation of the Inoculation Material and Method of Inoculation.**—The Lyons method is as follows: 40 grammes of infected muscle are rapidly dried at a temperature of 32° C., and are intimately mixed with 80 grammes of water. The mixture is divided into 12 equal parts, which are placed on separate flat plates. These plates are put into a thermostat for 6 hours to dry, 6 of the plates being exposed to a temperature of 100° C. in order to produce the weaker or first material, and the remaining six to one of 85° C., to obtain the stronger or second material. The inoculation is made in two stages, first with the weaker, and afterwards with the stronger material. The dried-up brown crust on the plates is used for the inoculation, and may be kept for a moderately long period. For the first inoculation, 0.1 gramme of the crust which was dried at a temperature of 100° C., is rubbed down in a sterilised dish with 5 grammes of water, so as to form the contents of a 5 grammes Pravaz's syringe. This syringe is manufactured according to Cornevin's designs by Lépine of Lyons, and its piston is graduated into ten equal parts, each indicating  $\frac{1}{2}$  gramme. The mixture is filtered

through damped clean linen, and the filtrate is drawn into the syringe. The quantity of material required for each animal is 0.5 c.c.; hence a syringe of suffices for 10 animals. The site of the first inoculation is on the lower surface of the tail, at about a foot from the tip. After the hair has been cut off, the skin is obliquely pierced with the trochar, which is supplied with the syringe, and the trochar is then pushed upwards for about 8 cm. between the skin and the bone. After its removal, the cannula of the syringe is introduced into the same wound, and the inoculation fluid, which is previously shaken up in the syringe, and thus uniformly distributed, is slowly injected. To prevent the escape of the fluid, the site of the puncture is compressed after the cannula has been removed, and the injected fluid is pressed farther up under the skin with the thumb. If there has been any hæmorrhage, we must wait until it stops before we inject, or we must choose another site. On the site of the inoculation we finally place an indiarubber band about 2 cm. broad, and retain it in position for four hours in order to prevent the flowing out of the injected material. It is stated that from 20 to 25 animals can be inoculated in this way in an hour. The animal is held by three assistants. Ten days after the first inoculation, the second one is made with the stronger material, and is carried out in the same way, except that its site is at about 8 inches from the tip of the tail. The spring or the end of winter is the most favourable time for the inoculation, which produces no secondary disease.

**Statistics of the Inoculation of Quarter-ill according to Arloing.**—In 1883 Cornevin inoculated with great success in *France*, according to the Lyons method, 125 animals, and also obtained very favourable results in 1884. According to Tisserand, inoculations were made in 1887 on 3,219 cattle, of which only 12 head (0.37 per cent.) died; and in 1888 on 2,086, of which only 3 (0.14 per cent.) died during the year. On the other hand, out of 4,000 non-inoculated animals, 50 (1.78 per cent.) died.

2. Following the example of Cornevin, Hess and Strebel made a large number of protective inoculations in *Switzerland*. Strebel states that over 2,000 cattle were inoculated up to the end of 1884, and were then kept during the whole summer on hills which were greatly infected by quarter-ill, with the result that the mortality of the inoculated animals was 28 times less than that of non-inoculated ones. He reports that 8,641 animals were inoculated in Switzerland during the years 1884–1888 with a loss of 15 (0.17 per cent.), and that 491 died of quarter-ill out of 21,000 (2.34 per cent.) that were not inoculated, and which had been kept under the same conditions as the other animals. Hence the mortality among the latter was 14 times more than among the former. He also says that the loss in 1889 among the non-inoculated animals in the Canton of Freiburg was  $12\frac{1}{2}$  times greater than that of the protected 6,616, and 8 times more in 1890. The proportionate average loss among the inoculated and non-inoculated animals for the seven years from 1884 to 1890 is consequently 1 to  $11\frac{1}{2}$ . He states that during 1885, in the Canton of Bern alone, 15,137 cattle were inoculated with very satisfactory results; for deaths from quarter-ill decreased very considerably in the infected districts. For instance,



in 2 villages in which this disease raged, the mortality of the protected animals was 8 times, and in another village 5 times less than that of the non-inoculated. According to Hess, 148,569 cattle were inoculated during the ten years from 1885 to 1894 in Bern with a loss of only 5 *pro mille*. With respect to the method of inoculation, Strebel likes best to inject in the region of the shoulder, especially in front of the shoulder-joint.

3. Sperrk inoculated during 1885 in *Austria* 925 animals with extremely good results; for although these cattle were afterwards sent to graze on notoriously infected summer pastures of the Tyrolean Alps, not one of them, so it is stated, died of quarter-ill. Three calves, however, died after the first inoculation. On the other hand, out of 6,387 non-inoculated animals, 107 died of the disease. During 1886, 2,140 young cattle were inoculated in Salzburg, and 3,820 in Tirol-Vorarlberg, with a loss of, respectively, 4 and 16; while at the same time, out of 9,160 non-inoculated animals in Salzburg, and 17,401 in Tirol-Vorarlberg, respectively, 86 and 330 died of quarter-ill. The mortality of the non-inoculated cattle, is consequently from 1 to 2 per cent., and that of the protected from 2 to 4 *pro mille*. The cost for inoculating each animal amounts on an average to from 6d. to 8½d. In Salzburg, 2,472 protected, and 3,561 non-inoculated cattle were depastured during 1887 on hills infected with quarter-ill, with the result that 6.3 per cent. of the non-inoculated cattle, and only 0.3 per cent. of the protected ones, died. According to this, the non-inoculated animals are twenty times more liable to become infected than the protected. In 1888, according to Suchanka, 0.16 per cent. of 1,773 protected cattle, and 1.74 per cent. of 3,036 non-inoculated cattle became affected. The losses during 1889 in the same place were 0.16 per cent. among 1,222 protected cattle, and 1.77 per cent. among 3,616 non-inoculated. According to Wildner, there was only one death from quarter-ill in Lower Austria among 1,011 protected animals. In *Hungary*, during the years 1891 to 1893, only 5 died out of 3,361 protected cattle.

4. In *Baden* during 1886 to 1894, only 3 died out of 3,567 protected animals. The consequence of these extraordinary good results is that in Baden an indemnity is paid for fatal cases of quarter-ill, if it can be proved that the animals had been inoculated.

**Kitt's single Inoculation** (*Munich method*).—Kitt observed during his inoculation experiments, that immunity can be produced in cattle and sheep by a single inoculation of vaccine which had been attenuated by exposure to a temperature of from 85° to 90° C. for 6 hours. This vaccine is about equal in effect to the second vaccine of Arloing, Cornevin and Thomas, and is harmless even if 10 times the ordinary dose be used. The local changes which appear after the inoculation are very slight and hardly noticeable. Kitt obtained a similar attenuated vaccine by means of the steam of boiling water. The results of Kitt's single inoculation are very favourable. From 1890 to 1892 in Salzburg, only 5 died out of 4,112 protected cattle, and 71 out of 5,286 non-inoculated cattle during the years 1890 and 1891; during 1892 in Innsbruck, only 13 out of 4,970 protected cattle; and during 1891 and 1892, in Vorarlberg, 23 out of 8,315 protected animals, and 303 out of 14,835 non-inoculated ones. In *Bavaria* there were no deaths among 167 protected cattle.

Only in Lower Austria, the experiments were so far unfavourable in 1893 that 12 animals died of quarter-ill in consequence of the inoculation, on which account the Lyons method was again adopted.

Owing to the great attention to detail required in the preparation of the vaccine from the dried flesh of animals that had suffered from quarter-ill, and the inequality of the preparations made from it, with consequent uncertainty as to the effect; Kitt has used lately the pure cultivations of the bacillus of quarter-ill for the inoculation material, especially cultures on agar-agar, which keep for a long time and which contain great numbers of spores. He gives the following details of his inoculation method :—

*"A pure fluid cultivation of the bacillus and spores of quarter-ill is kept in a test-tube closed with an indiarubber plug. If the tube remains plugged and is put into a cool dark room, the material will keep for several weeks. Consequently it is not necessary to use the material directly after it has been obtained. But if the plug be removed, the material must be used on the same day; for microbes which are in the air easily get into the fluid when the test-tube is opened, and the material becomes decomposed or mouldy. It also becomes spoiled by the influence of light. We use for the inoculation an ordinary Pravaz's syringe which holds 1, 5 or 10 grammes. The syringe should, first of all, be sterilised by boiling water, into which the cannula can be simply placed and the syringe repeatedly filled and emptied again. The use of a disinfectant solution is superfluous, and, besides, some of it might remain in the syringe and spoil the inoculation material. Shortly before making the inoculation, the test-tube is well shaken, so that the contents may be uniformly distributed, and the indiarubber plug is taken out and placed for the time being on the surface of a freshly-cut chip of wood. By holding the test-tube obliquely, its contents, which by this time have become turbid, approach the top of the tube, and can then be easily taken up into the syringe. If the syringe, instead of being completely filled, contains air-bubbles, it should be held in such a way that the air will come close to the cannula, and can then be injected into the test-tube, which is held perpendicularly with its mouth upwards, and the filling completed. Each of the cattle receives 1 c.c. of the inoculation material subcutaneously in the region of the elbow (above the four-headed extensor of the forearm). The skin of this part should be rubbed with a dry cloth, in order to remove dust and dirt; a fold of skin is taken up, and the cannula inserted. Having satisfied ourselves that the cannula has penetrated the subcutaneous cellular tissue, we inject 1 c.c. We should avoid puncturing the flesh, and should also take care that the needle does not go through the double fold of skin, in which case the inoculation material would escape on the other side. With a syringe which holds 5 or 10 grammes, and which has been filled, the regulating screw of the piston is adjusted to the first line of division, so that 1 c.c. may be injected, and after each inoculation the regulating screw is turned back to the corresponding line. If the manipulation be carried out in a clean manner, inoculations may be made out of one test-tube at several different periods during a day; but a test-tube which has been opened should not be kept longer than one day. The contents of those tubes which are not specially marked, are intended for the use of 20 head of cattle. After use, the test-tube, its remaining contents,*

and the indiarubber plug, are cleaned and sterilised in boiling water. In some cases, a slight swelling appears on the site of the inoculation, with or without a mild feverish reaction. It is advisable to repeat the inoculation after from 10 to 14 days on the elbow of the other side. The experiments which have been made up to the present, prove that a single inoculation confers immunity, which, however, is made doubly sure by a second one."

**Quarter-ill in Horses.**—In a place where quarter-ill was stationary among cattle, Ganter saw a horse die in the course of 24 hours, after having suffered from high fever, sweating, great depression of spirits, vomiting, and dyspnoea. *Post mortem* examination revealed a bloody, gelatinous, gas-containing infiltration on the neck and left thoracic wall, hæmorrhages in the muscles, and swelling of the spleen. The fluid in the gelatinous infiltrations of the throat contained the characteristic bacilli of quarter-ill, which fact was confirmed by Hafner.

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## INFLUENZA.

Equine influenza, or pink eye—Diseases of cattle resembling influenza—  
Influenza of man.

### **General Remarks on the Meaning of Influenza.**—

—The term, influenza, was originally applied in human medicine to an infective disease with catarrhal implication of various mucous membranes (*la grippe*). The word, which comes from Italian, is etymologically equivalent to "epizootic" or "distemper." In veterinary medicine it has long served as a collective name for all kinds of equine epizootics, the respective independence of which could not be proved by the light of the veterinary science of former days. People used the name "influenza" for every feverish, inflammatory malady of one or more of the internal organs which become either simultaneously or successively affective among a large number of horses. It was known as equine fever, typhoid fever, typhus, catarrhal fever, and erysipelatous fever. The extraordinary difference of the individual symptoms naturally led to the division of influenza into various forms, such as catarrhal, gastric, pectoral, erysipelatous, typhoid, rheumatic, and bilious influenza.

The first attempt to separate the most important infectious diseases which had passed under the name of influenza, was made by Falke. In his essay, *Influenza of the Horse*, which received a prize from the Royal Academy of Medicine of Brussels, he divided influenza into two principal diseases, which can be

diagnostically differentiated with certainty, and which are known at present as influenza, and equine contagious pleuropneumonia.

**History.**—According to Falke, influenza was mentioned as early as the time of Charlemagne by Charlemagne's private secretary Eginhard. Dieckerhoff states that it was probably known in the 4th and 5th centuries; in the 14th century, in Italy; in 1648, in Western Germany; and in 1711, in the Eastern provinces of Prussia. Heusinger tells us that it was fully described in 1729 by Löw (*Febris catarrhal, anni 1729 historia*) as an equine epizootic, which had spread over Italy, Austria, Poland, Wallachia, etc. It is also stated that cases of its having been transmitted to man had occurred. It was observed in 1732 by Gibson and others in London, and in different districts of England, where it raged epizootically in 1760, 1776, and 1803. The disease was widely disseminated at the end of the 18th and at the beginning of the 19th century, especially during 1805: From that time, influenza has continued to afflict horses in Europe and especially in Germany. The chief epidemics occurred in the years 1813 to 1815, 1825 to 1827, 1836, 1840, 1846, 1851, 1853, 1862, 1870 to 1873, 1881 to 1883, and 1890. Anker, who gave an admirable description of the influenza epizootic of 1826, in Switzerland, laid stress, even at that time, on the contagiousness of the disease, and stated that in his opinion a volatile infectious matter was the cause. The disease was subsequently described by Waldinger, Körber, Spinola, Hertwig, Hering, Köhne, and others, and in a very explicit manner by Falke in 1862. Influenza spread as an epizootic in 1872 to 1873 over the greater part of the United States, where it received the name of "pink eye" (French, *Fievre typhoïde*). The last great epizootic raged in Europe from 1881 to 1883, during which it spread over nearly the whole continent, and flourished chiefly in large towns, generally following the high roads of commerce. For instance, it was present in Berlin over two years, and in Munich for nearly a year and a half. From that time, Dieckerhoff, Schütz, Friedberger, Siedamgrotzky, Lustig, Vogel, and others have ably investigated and described the disease. This epizootic appeared in Northern Germany and Denmark during 1890, 1891, and 1892. In the Prussian army, 8,434 horses became affected in 1890; 2,497, in 1891; and 3,645, in 1892. In Copenhagen, 3,000 horses suffered from it in 1890 and 1891.

**Etiology.**—Influenza, which is as highly infectious as any other disease, can be produced only by infection, the nature of which has not yet been discovered. The disease can spread in a very short time among a large number of horses. It is transmitted, not in a disconnected and irregular manner, like equine contagious pleuro-pneumonia, but usually by successive infection. The contagium, which, without doubt, is exceedingly volatile, is probably absorbed from the inspired air and is contained in the air expired by affected and convalescent animals, and also in the excrements, as was shown experimentally by Friedberger. It appears to quickly lose its virulence outside the animal body. On the other hand, it seems to be sometimes preserved for a very long time in the animal body. According to the observations of Jensen and Clark, stallions which have had the disease may be able to transmit it to mares for months, and even for a year or two, by coition. Dieckerhoff succeeded in transmitting the disease to healthy animals by subcutaneous and intravenous injections of the blood of infected horses; but Friedberger and Arloing failed to do so. Horses are very susceptible to the contagium; their individual susceptibility being independent of age, sex, breed, stable management, feeding, etc. The infection generally takes place directly from horse to horse; though it may be carried by intermediate bearers, such as human beings, litter, harness, thermometers, etc. In many cases, one attack confers lifelong immunity; though a second infection or a relapse not unfrequently occurs. An epizootic invasion of influenza seems to last for one or two years. Towards the end of its duration, infections become rarer and milder, probably on account of a gradual attenuation of the contagium. At that time, the epizootic assumes a more or less sporadic character.

Influenza occurs also in asses and mules. In veterinary literature, we find a few accounts of supposed transmissions of equine influenza to man and dogs (Adam, Walther and Voss).

**Symptoms.**—After a *period of incubation* averaging from 4 to 7 days, the disease appears suddenly, and may attain its highest point of intensity within 24 hours. The organs of circulation, nervous centres, digestive and respiratory mucous membranes, eyes, and subcutis, become especially affected.

As *first symptoms*, we usually observe partial or entire loss of appetite, depression, and languor in movement. The internal

temperature, which is very high from the commencement, is characterised by a sudden rise up to  $42^{\circ}$  C. and even more, at which it remains with slight variations for from 3 to 6 days and then falls rather quickly to normal, often within 24 hours. At first, the frequency of the pulse is but little increased in comparison to the height of the temperature, and is usually only from 40 to 50 per minute; but later on it rises to from 60 to 70, and even to from 80 to 100, or more in fatal cases. It generally continues high for some time even after the temperature has fallen. The fever is characterised by unevenness in the distribution of the external temperature of the body. The fact of the internal temperature of apparently healthy horses which are among horses infected with influenza, being high, is of great diagnostic importance. In such infected stables, the temperature of the apparently healthy horses should be taken daily.

2. Great *nervous depression* co-exists with the fever. The animal holds its head down, and appears comatose, as if affected by sleepy staggers. Extreme muscular debility is frequently associated with this stupor. Tremors affect the body and limbs; the hind legs give way when walking; and paralysis of the hind-quarters appears in a few cases. The sclerotic is often of a yellow colour.

3. The *gastric symptoms* form an essential part of the disease. The oral mucous membrane is greatly congested, hot, dry, and often covered with mucus. There is sometimes difficulty in swallowing, when the inflammation of the oral mucous membrane spreads to the pharynx. The animal frequently yawns. Constipation is generally present at the beginning of the disease, and colic may occur. The fæces are formed into small hard balls and are covered with mucous membranous masses (proctitis). Later on, the constipation often changes into diarrhoea with considerable tenesmus. The fæces are then of a thin, pulpy, and even fluid consistence, and sometimes have a foetid smell. The peristaltic action of the bowels is usually suppressed, and the hind part of the body is here and there painful on pressure. The urine at the beginning of the attack is alkaline in spite of the high fever, and becomes acid only when the intestinal affection becomes developed. Although it is not often albuminous, it frequently contains large quantities of desquamated epithelium of the bladder. Sometimes the animal stales very frequently (slight catarrhal cystitis).

4. A more or less severe affection of the *eyes* is a rather

constant and is a very characteristic symptom of influenza. At first it consists chiefly of a catarrhal, and later on of a phlegmonous conjunctivitis with considerable swelling of the eyelids (chemosis), which may be followed by parenchymatous keratitis and even by an exudative or hæmorrhagic iritis. Generally both eyes become affected. The first symptoms are tears, intolerance of light, great congestion of the mucous membrane of the eyelids, and contraction of the pupil. The eyelids swell considerably, are hot, painful to the touch, and are kept persistently closed. The mucous membrane of the eyelids is glassy, œdematous, and often projects outwards between the eyelids in the form of an orange-coloured protuberance. The conjunctiva scleræ frequently forms a grey, translucent, raised, and tumid ring in the neighbourhood of the cornea. A grey, muco-purulent secretion accumulates between the eyeball and the eyelids, and the eyeball becomes very sensitive to pressure. The cornea at the beginning of the keratitis has a greasy lustre, is iridescent, smoky, and appears as if covered with dust. Later on, a blue or milky opacity forms over its surface. It is considerably injected with blood at its edge; the iris becomes swollen and assumes a yellow colour; and exudate and blood are sometimes seen in the anterior chamber of the eye. Often these inflammatory changes of the eye disappear in a strikingly rapid manner.

5. During the further process of the disease, *swellings* appear on the extremities, sheath, epigastrium, and lower part of the chest. We may conclude that these swellings are due to œdema caused by passive congestion; because they are painless, cool, and doughy, and because they appear when the action of the heart becomes weak, during the later stages of the disease. Less frequently, the swellings are of an inflammatory (phlegmonous or erysipelatous) character. The swelling of the extremities causes the gait to be stiff and straddling. The swellings often completely disappear in a very short time on the approach of recovery. Inflammation of the sheaths of the tendons is sometimes observed. In a few cases, an eruption of wheals, outbreaks of sweat, and loss of hair have been seen.

6. The *respiratory mucous membrane* suffers from slight catarrh. At first there is a serous, and, later on, a muco-purulent discharge from the nose; slight swelling of the submaxillary glands; moderate acceleration of respiration; cough; and congestion of the mucous membrane of the vagina. As a rule, the animal becomes a good deal emaciated

during the course of the disease ; and pregnant mares generally abort.

**Course and Complications.**—The average duration of the disease is from 6 to 10 days ; though severe cases may run on for 2 or 3 weeks, and very mild cases may recover in from 3 to 6 days. As soon as the temperature falls, the appetite usually improves ; the peristaltic action of the bowels becomes more brisk ; the spirits brighter ; the swellings smaller ; and recovery becomes complete in from 8 to 14 days. In a few cases, complications set in, especially when the horse has been worked hard during the initial stage of the disease, or during convalescence. The complications are as follows :—

1. *Pneumonia* becomes developed with inflammation of the respiratory mucous membrane, as catarrh of the lungs, with tendency to necrosis of the lungs, or as croupy pneumonia ; and may secondarily cause pleuritis. The rate of respiration and of the pulse becomes greatly increased ; the temperature rises to a high degree ; and the general health suffers considerably. In rare cases, the mucous membrane of the larynx becomes affected with a phlegmonous swelling, which manifests itself by severe dyspnœa and by the production of sounds due to constriction of the lumen of the part.

2. *Cardiac debility*, which is manifested by palpitation of the heart ; very frequent, small, and finally imperceptible pulse ; dyspnœa ; and passive congestion of the mucous membranes.

3. Grave *cerebral symptoms* (lepto-meningitis and cerebral apoplexy) with unconsciousness and finally paralysis of the brain ; spinal paralysis (paralysis of the loins) ; monophlegia (paralysis of the facial) ; and amaurosis (atrophy of the papillæ).

4. Severe *gastro-intestinal inflammation* and *profuse diarrhœa* ending in death.

5. *Laminitis* caused by spreading of the inflammatory swelling of the skin to the sensitive laminæ, or by long standing.

6. *Petechial fever*, *inflammation of the kidneys*, *peritonitis*, *inflammation of the testicles*, *rupture of the spleen*, *uncontrollable diarrhœa*, *tendo-vaginitis*, *abscesses*, *muscular spasms*, and *phlebitis*.



**Anatomy.**—The principal changes of influenza are met with in the *organs of digestion*. The mucous membrane of the pyloric portion of the stomach and intestines (especially of the large intestine, including the rectum) is hyperæmic, swollen, and infiltrated with slight hæmorrhages; and its epithelium is desquamated. The submucosa is yellow and gelatinously infiltrated to a high degree, so that the mucous membrane forms thick, translucent elevations which have a glassy lustre, and which may attain the thickness of a few centimetres. On being opened they are found to contain a fluid which coagulates in gelatinous lumps. Peyer's patches are enlarged, and present medullary swellings, especially in the neighbourhood of the ileo-cæcal valve. The mucous membrane of the oral cavity and sometimes that of the pharynx show similar changes.

The mucous membrane of the upper *air passages* is catarrhally congested and swollen; and in rare cases the mucous membrane of the larynx is phlegmonously inflamed (gelatinous infiltration of the submucosa), which condition is also found in the subcutis when inflammatory swellings appear on the skin. Schütz states that in the *brain* and *spinal cord* the arachnoideal spaces, particularly on the basilar surface, are filled with a fluid which is generally clear, and which contains leucocytes. The plexuses of the veins may suffer from cloudy swellings. In one case, the lateral ventricles contained a large quantity (20 c.c.) of fluid.

The chief *general changes* are as follows: cloudy swelling of the heart, kidneys, liver, spleen, muscles, etc.; slight swelling of the spleen; small hæmorrhages in the intestines, under the serous membranes, and in the lungs, eyes, and brain; gelatinous infiltration of the renal connective tissue, and mesentery; swelling of the lymph glands; yellow, serous transudations in the cavities of the body; and an imperfectly coagulated condition of the blood.

**Prognosis.**—Influenza is a mild equine disease. The mortality, which varies at different times and in different places, is on an average from 0.5 to 4 per cent.; being greatest at the beginning of an epizootic invasion, and least towards its end. Dieckerhoff saw a loss of 4 per cent. in 1,700 infected horses; Aureggio, one of 3 per cent. among 800 horses; Friedberger, one of 9 per cent.; and Siedamgrotzky, one of 10 per cent. The average yearly mortality among the horses of the Prussian army during the years 1886–1893 was only 0.5 per cent.

Friis puts the mortality during an epizootic in Copenhagen (1890) at 1 per cent. It is stated that in 1872 in Philadelphia, 7 per cent. died among 30,000 infected horses.

**Differential Diagnosis.**—Influenza is distinguished from other infectious diseases by its epizootic occurrence, rapid dissemination over large tracts of country, great contagiousness, benign character, speedy development, and sudden appearance of the symptoms. It can be separated from equine contagious pleuro-pneumonia by the special implication of the digestive organs and of the eyes; by the severe nervous symptoms; and by the slight participation of the organs of respiration, which in pleuro-pneumonia are the chief seat of the disease from the commencement of the attack. The differentiation between pleuro-pneumonia and influenza can be difficult only at the beginning, when merely general symptoms, such as fever, loss of appetite, and weakness are present. It must be mentioned that these two diseases may simultaneously affect a horse. When the skin is greatly swollen, influenza may somewhat resemble petechial fever, from which it can, however, very soon be distinguished by the absence of petechiæ, by the mildness of its course, and its greater contagiousness.

**Therapeutics.**—On account of the mild and typical course of influenza, medical treatment is generally superfluous; nursing, and attention to diet and ventilation being, in such cases, sufficient for our purpose. When practicable, and when the weather is favourable, it is best to have the sick animal in the open air; keeping him, of course, at a distance from healthy horses, and close to a stall (or hovel) of his own. Some recommend giving large quantities of milk (12 to 15 litres *pro die*). The temperature may be reduced, and the peristaltic action of the bowels may be stimulated by enemas of cold water.

Opinions differ with regard to the benefit of febrifuges in influenza. Friedberger, Luchau, and others advocate the administration of antifebrin, even at the beginning of the fever, in two or three doses of from 30 to 45 grammes, which, as a rule, rapidly reduce the temperature, and favourably influence the course of the disease.

Medicines are necessary in severe and complicated cases. We may combat symptomatically the cardiac weakness with alcohol, wine, camphor, digitalis, strophanthus, caffeine,

atropine, hyoscine, and veratrine; the gastric affections, with alkalies (the sulphate and bicarbonate of soda, and common salt); the inflammation of the eyes, with atropine; the swelling of the skin, with inunctions of camphor; and the high, persistent temperature, with antifebrin (100 grammes *pro die* in three doses). The healthy animals should be separated from the infected, and a thorough disinfection of the stable should be made.

Veterinary police regulations against the disease have been advocated formerly by Spinola, and recently by Lustig and others. We, however, share the opinion of Dieckerhoff, that severe regulations cannot be carried out without serious disadvantage to traffic, and that they are also not very necessary, considering the benign course of the disease. As proposed by Lustig, it might be well to introduce compulsory notification in every case of influenza, in order to direct increased attention to this epizootic, and to its infective character. Public instruction might also be given on this subject. In Prussia an edict of the Minister of Agriculture (December 15th, 1889) orders that in the event of an outbreak of influenza, or of equine contagious pleuro-pneumonia occurring, the official veterinary surgeons and the police authorities, should send in reports, which should be published in the official papers, and communicated to the directors of breeding establishments, and to the military authorities.

**Diseases of Cattle resembling Influenza.**—Many have observed among cattle, peculiar epizootic morbid conditions which resembled equine influenza. Bräuer found in 12 cattle, which were in 2 sheds of 6 each, catarrhal symptoms, great depression, inability to bear fatigue, giving way in the hind-quarters, dizziness, high internal temperature ( $41.5^{\circ}$  to  $42^{\circ}$  C.), and dark orange colour of the nasal mucous membrane and of the conjunctiva. Mosses saw in several milk-cows of a farm loss of appetite, great weakness, decrease of milk, high temperature ( $39^{\circ}$  to  $40.9^{\circ}$  C.), dark-coloured mucilaginous excrements, swelling and yellow colour of the conjunctiva, copious lachrymation, opacity of the cornea, bloody exudate in the anterior chamber of the eye, yellow colour of the oral mucous membrane, and painful tumours on the skin. The disease, which ended in recovery, lasted from 8 to 14 days. Spinka reports a similar case. Harms states that in a cattle disease, which he termed influenza, the following symptoms were constantly present: gastro-enteritis, synovitis, mastitis, which was frequently complicated with vaginitis, bronchitis, and conjunctivitis, with occasional inflammation of the external and internal parts of the eye. Janson states that an epizootic, which was confused by many with rinderpest, raged in Japan during 1890 and 1893. This epizootic took the form of a very acute, feverish, and contagious affection of all the mucous

membranes, especially of the respiratory tract. Its duration was generally only 2 or 3 days, and usually ended in recovery. The animals were depressed; could get up off the ground only with difficulty; trembled; ground their teeth; had feverish rigors ( $40^{\circ}$  to  $42^{\circ}$  C.); the conjunctiva was dark-red and swollen; the eyes suffered from photophobia and lachrymation; a watery discharge issued from the nostrils; and there was difficulty in breathing, considerable salivation, and at first constipation, with the fæces covered with mucus, and later on diarrhœa. At the same time painful swellings affected the joints, especially the hock, and, in very severe cases, the skin suffered from an extensive attack of emphysema.

**Influenza of Man.**—The acute infective disease of man, which is termed influenza, or *la grippe*, and which, according to the recent researches of Pfeiffer and Canon, is caused by a small bacillus, has been discussed by medical men in connection with equine influenza. The epidemic outbreak in 1891 of *la grippe*, which is a disease that has been known for a long time, has directed increased scientific attention to it. The bacilli of human influenza, which are contained chiefly in the sputum, seem to enter the body of man principally by the air passages. The disease manifests itself by high temperature, mental depression, rhinitis, pharyngitis, laryngitis, bronchitis, rheumatic affections of the muscles and tendons, etc. These phenomena may be associated with extremely varied complications, such as inflammation of the lungs, inflammation of the brain, myocarditis, and other sequelæ. Pfeiffer states that the disease can be transmitted by inoculation to monkeys and rabbits. We have no proof that human influenza is identical with equine influenza. It is even improbable that the two diseases are in any way connected together. In our opinion they are entirely different infective diseases, which are in no respect etiologically related to one another. The respective differences between the course of the outbreaks, the morbid phenomena, and the fact that the transmission of the virus from man to horse, or *vice versa*, has never been proved to have taken place, are strong arguments against the identity of the two diseases. The same remark holds good with respect to any supposed identity between human influenza and canine distemper.

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#### EQUINE CONTAGIOUS PLEURO-PNEUMONIA.

**Nature, Occurrence, and Pathogenesis.**—Pleuro-pneumonia is an infective inflammation of the lungs, in the course of which the pleuræ (pleuro-pneumonia), and large organs of the body, such as the heart and kidneys, participate in the morbid process. Its infective matter has not yet been found with certainty. The respective statements of bacteriologists, to which we shall refer later on, differ greatly from each other. The disease occurs enzootically, principally in

places where large numbers of horses are kept ; for instance, in cavalry regiments, army depôts, breeding establishments, and stables of horse dealers ; and is more frequent in towns than in the country. Horses from 5 to 10 years appear to be more susceptible to it than older horses, some of which, perhaps, have already had the disease. One attack generally gives immunity for several years, and sometimes even for life, although exceptions are not uncommon. Horses are not so susceptible to pleuro-pneumonia as to influenza. Rust states that in a thoroughly infected regiment of 705 horses, only 245 took the disease ; 400 proved to be immune ; and 60 had had the disease. Horses in good health are less susceptible than those with a weak constitution. Over-exertion, chill, catarrh of the lungs, and bad ventilation in the stable are predisposing agents. Consequently the disease is, as a rule, more frequent in winter than in summer.

The contagium can be directly transmitted from animal to animal by the respired air. It can, for instance, be introduced by remounts and working horses, and is transmitted from horse to horse. The infection, however, is carried usually by intermediate bearers, such as human beings (especially veterinary surgeons and grooms), healthy horses, dung, clothes, forage, strange stables, and even dogs. The infected horses of the civil population of garrison towns are a continual source of infection for army horses. Convalescent horses are particularly dangerous ; because they can carry about in their bodies the infectious matter, frequently, as it seems, for weeks (dead lung-deposits). It has not yet been positively proved whether a transmission of the disease, without direct or indirect infection, by means of a miasma, that is, by a spontaneous development of the contagium outside the animal body, may occur.

The frequency of pleuro-pneumonia in badly ventilated and insufficiently drained stables, and the fact that not uncommonly sporadic cases may be observed in a stable throughout the whole year, have led to the supposition that the infective matter may be preserved in a stable as a stable-miasma for a long time, and may thus cause the disease to become stationary. On the other hand, the occurrence of such cases may be explained by the supposition that a direct or indirect infection is transmitted by one or more infected horses which are slowly recovering from the disease, and which are apparently healthy ; or by direct or indirect infection from outside, which is always possible, considering the wide dissemination of the disease. The question

mooted by Peters, of the connection between pleuro-pneumonia and variations of surface-water, requires to be further investigated. The appearance of pleuro-pneumonia in a stable is different from that of influenza, which attacks horses in turn. The order in pleuro-pneumonia, on the contrary, is quite arbitrary, as if it was regulated by intermediate bearers. The army horses which occupy corner stalls are most frequently and most severely attacked, probably on account of inferior ventilation.

The course of the disease is generally most rapid at the commencement of an outbreak. A case has been known of 42 horses of one squadron becoming affected within 2 days, and another case of the disease spreading within 3 days through an entire military stable. Frequently the horses of a whole regiment become infected within a fortnight. On an average, the epizootic disappears 5 or 6 weeks after the occurrence of the last case, provided that no fresh case is introduced. Subsequently, the course of the disease generally becomes chronic and indolent, and may remain stationary in some stables, if the horses in them have become chronically affected with lung disease (cavities of the lungs), and, on that account, maintain the supply of the infection; or the disease may be introduced again and again from outside by apparently healthy horses (in the army, especially by remounts).

The respiratory organs are the chief places of entrance of the contagium, which probably gets first into the lungs by inspiration, and from thence into the blood. It is not improbable that the infection may also take place from the digestive canal.

The period of incubation is generally given as from 5 to 10 days; although frequently it may be only from 1 to 4 days, or may sometimes extend to 10 days or a fortnight. It is very difficult to fix its exact duration; because it is impossible to find out the precise time the infection takes place. Consequently, many of the statements made as to its length are contradictory. In no case should we, as was frequently done, calculate the period from the time the healthy horse has been placed in an infected stable to the appearance of the first symptom. We need hardly say that the period of incubation is that interval which lies between the absorption of the infective matter into the body and the appearance of the first symptom. The time at which the absorption of the contagium takes place cannot, as a rule, be determined.

**Bacteriology.**—1. Klebs, Ebert, Koch, Friedländer, Fränkel, Zäslein, Salvioli, and others have found micro-organisms, which are the exciters of inflammation in human lungs, in the form of streptococci, and often diplococci, and which are surrounded with a capsule.

2. In cases of inflammation of the lungs of horses, Peterlein, Perroncito, Brazzola, and Mendelsohn were the first to find in the inflamed parts of the lungs streptococci, the cocci of which were separated from each other by a bright zone; and diplococci (Peterlein). Perroncito describes as "*bacterium pneumoniae crouposa equi*," large cocci which are almost spherical or ovoid, single or in twos (diplococci), sometimes also in threes, and rarely connected in chains (streptococci), and which are often surrounded by a bright, gelatinous ring. Perroncito distinguishes these cocci from those of man by the circumstance that they are always pathogenic in guinea-pigs and rabbits, and that they cannot be stained with the solutions which are used for the staining of the capsule of the cocci of man. In all these experiments the pathogenic character of the cocci in question has been proved neither by pure cultivations nor by inoculation.

3. Lustig prepared in 1885 pure cultures of a micro-organism which he considers to be the contagium of equine contagious pleuro-pneumonia, and with which he made inoculations later on. He obtained in his bacteriological researches six different cultivations, of which he considers the sixth, a yellow cultivation, to be the exciter of this disease. The cultivation grows chiefly on the surface, but only to a small extent, and also grows in the track and at the bottom of the puncture in gelatine. These stab-cultivations have almost the form of a nail. The cultivations consist of very minute, ovoid bacilli, which can be stained with Gram's solution. They are stained easiest in a saturated solution of dahlia. Lustig obtained the following results from inoculations with these cultivations: 9 horses that were inoculated in the thoracic cavity became affected with pleuro-pneumonia, with the result that 2 died and 7 recovered. No. 6 culture was able to be obtained out of the pleuritic exudate in one of the horses while it was alive, but which subsequently died. Four horses which had been inoculated in the thoracic cavity were afterwards exposed with five horses which had not been similarly inoculated, to the natural infection, with the result that the former remained healthy and all of the latter became attacked by the disease. Schütz and Lüpke have not been able to find the micro-organism observed by Lustig, and have declared that they do not consider it to be specific.

4. Schütz believes that the contagium of equine pleuro-pneumonia consists of small oval bacteria which are usually grouped together as diplo-bacteria, and which have been formed by the transverse division of individual bacteria. These bacteria can be stained with methylene blue and all basic aniline colours, and are characterised by the fact that they become decolourised when Gram's method is used. We can sometimes recognise around them a ring which in some cases can be stained, but cannot in others. Stab-cultures on meat-water-peptone-gelatin produce small, white, spherical tufts which do not liquefy the gelatine and which do not spread over its surface. Cultivations in broth show white flakes at the bottom of the tube, and on agar-agar they form small, opaque, grey tufts. The pure cultivations can be transmitted

to mice, rabbits, pigeons, and guinea-pigs, but not to pigs or fowl. The inoculation produces in mice a septicæmia which generally ends in death in 24 to 48 hours. According to Schütz, the cultivations can be transmitted effectively to horses, and when injected directly into the lungs by means of a Pravaz's syringe they produce contagious pleuro-pneumonia, which has the same symptoms and course as those observed in natural cases of this disease. The essential changes shown on *post mortem* examination were multiple gangrenous patches in the lungs with parenchymatous degeneration of the most important organs. The characteristic bacteria were found in the body of the inoculated animals. According to Schütz, bacteria of contagious pleuro-pneumonia are found most numerous in the lungs and in the exuded matter of the pleuræ, and are also met with in the nasal discharge and in expired air (Rust). The development of the bacteria ceases at a temperature of 10° C., and evidently is favoured by moist heat, such as that of a manure heap. Nothing positive is known respecting the duration of life of the bacteria outside the animal body. They are usually supposed not to be able to live longer than 6 weeks in the animal body. The contagium may, however, continue active for a very long time, especially in encysted deposits in the lungs.

Baumgarten and Hell oppose the view that Schütz's bacteria are specific, while Rust and Fiedler support it. Baumgarten considers that this subject is not fully explained. Hell maintains that with our present means of investigation, the bacteria of contagious pleuro-pneumonia cannot be differentiated from those of the streptococci of pus, and from the streptococci of erysipelas. In fact, Hell believes that the ovoid cocci and diplococci of Schütz have a pathogenic effect in the infection of horses with pleuro-pneumonia; but as we have no positive proof of their being specific, he maintains that we are justified in supposing that these ubiquitous micro-organisms have only an injurious influence on the course of pleuro-pneumonia, and that they contribute to the production of the secondary affections, especially of the necrotic foci of the pleuritis. Hell further states that protective inoculation with Schütz's bacteria, which at first promised good results, has not fulfilled the expectations formed of it. Fiedler, on the other hand, has obtained the same bacteriological results and has arrived at the same conclusions as Schütz. He also states that he has experimentally produced pleuro-pneumonia in a horse by inoculation of cultivations of these bacteria.

It therefore appears that the bacteriology of equine contagious pleuro-pneumonia is still an open question.

**Anatomical Changes.**—In the lobular form of pleuro-pneumonia, which furnishes the largest number of subjects for a *post mortem* examination, we find a multiple hæmorrhagic, gangrenous pneumonia with secondary pleuritis and parenchymatous degeneration of the chief organs of the body.

1. We find portions of the *lungs*, of greater or less size, thickened, hepatized, and devoid of air, especially near the root of the lungs and in the lower sections; and bright foci, which are distinctly defined from the neighbouring tissues, sprinkled



over the dark-red sections of the hepatised parts. Usually, several of these foci are present, though sometimes only one may be met with. They vary in size from a pin to a child's head. At first they are very small; of a greyish-red colour (hæmorrhagic foci), and are surrounded by a whitish zone of emigrated leucocytes (limited reactionary inflammation).\* Later on, they become yellowish, resemble tinder in appearance (dry necrosis), and then take the form of cavities, which may be as small as a pea or as large as a hen's egg, and which contain necrotic lung tissue. These cavities are surrounded by a smooth capsule. There are other foci which contain greasy, fœtid, watery pus (gangrene of the lungs), by reason of the necrotic parts of the lung undergoing watery disintegration, in consequence of the admittance of air. We also find in the lungs suppurating foci, which are generally of rather a large size, and which contain a whitish pus, mixed with dead lung tissue (suppurative demarcation of the necrotic parts). It may often happen that none of the foci which we have just described are to be seen in the lungs; although, during life, distinct symptoms of such a localised affection may have been present. In these cases, we must assume that absorption of the necrotic tissue has occurred. The remaining tissue of the lungs is, more or less, hyperæmic and œdematous.

2. The *pleuræ* show signs of a diffuse, exudative pleuritis, the starting-point of which, in the large majority of cases, is from necrotic deposits which are situated on the periphery of the lungs. This pleuritis may, however, appear without these foci being present. The contents of a necrotic deposit in the lungs rarely breaks through into the pleural cavity. Both the visceral and costal layers of the *pleuræ* are congested diffusely and in spots, and are infiltrated with hæmorrhages; are dull and rough; and are often covered with soft, red granulations. Over them we find soft, yellow layers, which are partly membranous and partly coagulated in a reticular manner, and which can usually be easily removed. The cavities of the *pleuræ* generally contain a considerable quantity of fluid, which may amount to 7 gallons or more. It is usually turbid; is of an orange, grey-red, brown-red, or dirty-greyish colour; and is generally mixed with numerous yellow flakes which form a sediment when the fluid has been left to stand in a glass. The pleuritic exudate sometimes consists of pure pus (empyema),

\* Inflammation which is caused by reaction to external influences, and which leads to demarcation or casting off of dead portions of tissue. — TR.

and less frequently of blood (hæmatothorax). Pneumothorax is very rarely present. The exudate compresses the lungs and pushes them away from the thoracic walls. In consequence of the exuded fluid becoming organised, the lungs become adherent to the costal walls and diaphragm, and fibrous, villous growths develop on the pleuræ.

3. The *other organs of the body* are in a state of parenchymatous inflammation and fatty degeneration. The *muscular tissue of the heart* is, as a rule, brownish-grey in colour, soft, and suffers from cloudy swelling. In severe cases it shows well-marked fatty degeneration, is of a clay colour, and is occasionally infiltrated with a large number of small, yellowish-white foci. The *liver* is enlarged, of a clay colour or sometimes icteric, and presents signs of fatty degeneration. The *spleen* is flaccid, and its pulp increased and often infiltrated with hæmorrhages. The *kidneys* are swollen, brittle, and sometimes show numerous hæmorrhagic foci. The *lymph glands*, especially the bronchial and mediastinal glands, are enlarged, softened, and exhibit a grey-red colour on section. The *muscles of the body* are soft, and of a yellowish-brown colour. We may frequently find small *hæmorrhages* under the serous membranes, and slight endocarditis. The *blood* suffers from less change than any of the tissues.

4. The *mucous membrane of the stomach and intestines* is frequently hyperæmic, swollen, infiltrated with hæmorrhages, and sometimes even ulcerated. Its epithelium is desquamated, and its lymphatic glands are swollen, and often fall out. The bronchial mucous membrane is also swollen and inflamed.

The anatomical changes of the less frequent lobar form are those of croupy pneumonia.

**Symptoms.**—The symptoms are of such a varied character that it is quite impossible to give an exact description of them. We shall first mention the most frequently observed symptoms, and then put the complications under a separate heading. Pleuro-pneumonia appears rather suddenly, or is preceded by premonitory symptoms, such as general faintness, loss of appetite,\* cough, etc. The first regular symptom is a rapidly-increasing temperature, which generally rises to 40° or 41° C., and which frequently begins with rigors. It often rises 3° C. within a few hours. The rate of the pulse increases to from 50 to 70 beats a minute, and often, later on, to 80 or 100, or even more.

\* McFadyean remarks that, contrary to the usual opinion, the appetite, as a rule, is fairly well sustained in this disease.

On the whole, the pulse is more frequent in pleuro-pneumonia than in influenza. At the same time, during the later stages of the former disease, it is very small, and there is severe palpitation of the heart. We have also depression and muscular weakness, though not to such a high degree as in influenza; and the conjunctiva and other visible mucous membranes are congested, and are deeply tinged with yellow. There is almost entire loss of appetite. The character of the lung affection may vary in cases of equine contagious pleuro-pneumonia, which presents the two following entirely different clinical aspects.

1. A *lobular* inflammation of the lungs with tendency to gangrene of the lungs and pleuritis.

2. A *lobar* inflammation of the lungs, which is of a mild character, and which has no tendency to gangrene and pleuritis.

#### LOBULAR-PNEUMONIC FORM OF EQUINE PLEURO-PNEUMONIA.

This affection of the lungs is localised and manifests itself at first by cough, and accelerated and difficult breathing, as well as by high fever and other general symptoms. If the pneumonic foci are very small, no dulness of the lungs will be found on percussion, and auscultation will reveal nothing abnormal beyond the fact that the vesicular breathing is strong and shrill. Pleuro-pneumonia in such cases is difficult to diagnose, and may be easily mistaken for influenza. If several pneumonic deposits be close together, lobular, irregular dulness may often be found on percussion, especially in the antero-inferior parts of the lungs, such as directly behind the shoulder, and particularly on the left side near the heart; and weakness of the vesicular respiratory murmur. The presence of this lobular pneumonia can generally be ascertained after the third day of the attack. At the same time we may observe the appearance of a rusty-yellow nasal discharge, which sometimes dries into a brownish-yellow crust near the nostrils. The cough becomes weaker and the difficulty of breathing increases.

The extension of the inflammatory process to the pleuræ is at first manifested by the dry friction sound, and by the highly sensitive condition of the affected places on the thoracic walls. Respiration becomes abdominal. With the accumulation of a pleuritic exudate in the thoracic cavity, the dulness becomes more extensive; it assumes a marked horizontal direction; and may extend over two-thirds or more of the thoracic cavity. On auscultation in such cases, we find that there is no

vesicular respiratory sound, or that there is bronchial respiration on the boundary of the dulness. The change of lobular-pneumonic places into necrotic cavities is shown externally on percussion by a tympanitic sound, or by a cracked-pot sound (*olla rupta*). The same sounds are heard with localised pleuritic exudates. With the increase of the pleuritic exudate, respiration becomes extremely difficult, and the horse while trying to breathe, widely distends his nostrils, which, so to speak, take the form of trumpets. The course of the fever is sometimes typical; at other times, atypical.

If the disease runs a favourable course, improvement takes place after the above described symptoms have lasted for 6 or 8 days. This improvement is generally preceded by a critical polyuria and rapid lowering of the temperature. The recovery is complete after a period of convalescence, which lasts from 2 to 4 weeks. Such a rapid and favourable course can be expected only when the animal has not suffered from pleuritis with abundant exudation. In severe cases, the animal may die during the first week. Death is caused chiefly by a series of grave complications.

#### LOBAR-PNEUMONIC FORM OF EQUINE PLEURO-PNEUMONIA.

This, which is the milder form of pleuro-pneumonia, is characterised by its typical course and large areas of dulness. Exudative pleuritis is rare in it. The inflammation of the lungs begins also on the antero-inferior parts of the lungs, and extends rapidly over large sections of these organs. In the majority of cases the inflammation is unilateral; that of the left side occurring about twice as often as that of the right side. Friedberger found only 8 patients afflicted with bilateral pneumonia out of 70 cases which he had examined in order to decide this point. In the initial stage, percussion of the thorax produces a tympanitic sound, which is seldom quite pure, and is generally somewhat dull. The second stage, namely, that of hepatisation, is distinguished by a dull percussion sound with the feeling of considerable resistance to the hammer. Tympanitic sounds are also noticed on the boundary of the dull sound. In the third stage, we again find the tympanitic sound. This typical character of the percussion sound is not met with in abortive cases; because, owing to the shortness of the course of the disease, percussion reveals no changes, or only trifling ones. At the beginning of the attack, auscultation of the lungs,

and especially of the boundary zones, reveals crackling râles ; in the second stage we hear, chiefly during expiration, bronchial breathing ; and in the third stage, moist râles. Auscultation of the healthy parts of the lungs manifests increased and frequently a rough vesicular respiratory murmur.

The respiration, which is generally between 20 and 60 a minute, is accelerated and difficult, in proportion to the height of the fever and to the extent of the inflammation of the lungs ; and is chiefly costal. The nostrils are widely dilated and sometimes give out a peculiar flapping sound, which Dieckerhoff believes to be due to temporary adhesion of the mucous membrane of the false nostril to that of the opposite side. The expired air is abnormally hot. The sufferer either persistently stands up with the fore legs as a rule wide apart, or lies down only on the affected side, and is careful to avoid any movement. We may hear at the same time a shallow, short, and painful cough, which is often entirely suppressed. At the beginning of the attack, in about 25 per cent. of all the cases, especially in the severe ones, and in those in which the pneumonia assumes a well-marked hæmorrhagic character, we find a nasal discharge which may vary in colour from saffron-yellow to dirty-yellow, and which, on drying, forms yellow crusts about the nostrils.

The ordinary course of lobar pleuro-pneumonia is typical in that the stages of engorgement, hepatisation, and absorption, follow each other at equal intervals of time. The course of the abnormally high temperature is also typical. The disease usually reaches its crisis on the fifth or sixth day, after which resolution begins. The dulness often disappears in a surprisingly short time ; rhonchi take the place of bronchial respiration ; the breathing becomes more tranquil ; and the cough looser. Recovery, as a rule, takes place in from 8 to 14 days.

**Complications of Equine contagious Pleuro-pneumonia.**—During the course of this specific pleuro-pneumonia, besides lobular or lobar pneumonia and pleuritis, we may have a series of symptoms which, with a combination of pneumonia and pleuritis or by themselves, may present the aspect of the disease we are considering. The heart, membranes of the brain, and kidneys are sometimes so exclusively the seat of the disease, that the lungs may be unaffected. According to the statistics collected on one occasion, inflammation of the lungs and pleuræ was absent in 27 cases out of 117. The complications are as follows :—

1. A severe *cardiac affection* shown by parenchymatous inflammation of the myocardium, which is one of the most common and most dangerous complications of pleuro-pneumonia, and which manifests itself by a very frequent and small pulse, that finally becomes imperceptible; palpitation of the heart; extreme weakness, and general symptoms of congestion, especially in the lungs (dyspnœa).

2. Grave *gastric symptoms*, such as severe colic, diarrhœa, and bloody fœces.

3. A *hæmorrhagic or catarrhal (parenchymatous) nephritis*, which is characterised by hæmaturia; presence of casts of blood and of exudates in the urine; great weakness of the hind legs; anasarca (parenchymatous nephritis); and increasing general anæmia. We have seen fatal cases of this complication, in which the inflammatory lesions in the lungs had completely healed.

4. *Septicæmia* and *pyæmia* following the formation of serous and suppurating foci in the lungs.

5. *Cerebral convulsions* and *symptoms of a leptomeningitis* in consequence of the contagium of pleuro-pneumonia attacking the cerebral membranes (the so-called nervous form of pleuro-pneumonia). These cerebral symptoms are sometimes the first and only symptoms of pleuro-pneumonia. As a rule, death rapidly ensues from paralysis of the brain.

During the progress of the disease, we may also observe copious hæmorrhage of the lungs; hæmorrhage of the kidneys; intermitting inflammation of the tendons and their sheaths; laminitis; internal inflammation of the eyes (iritis exudativa et hæmorrhagica); roaring (frequently in 10 per cent. of the affected horses); epileptiform convulsions; muscular spasm in the region of the facialis and trigeminus; muscular rheumatism; arthritis; rupture of the spleen; weakness of the loins; paralysis in the region of the ischiadicus; paralysis of the rectum, tail, bladder, and penis (in very rare cases); chronic endocarditis with valvular lesions; chronic pericarditis; broken wind; anasarca (a symptom of parenchymatous nephritis); pharyngitis; phthisis pulmonum; general mal-nutrition; general falling out of the hair; and paralysis of the pharynx. During the course or towards the end of an attack of pleuro-pneumonia, the affected animal may become seized with influenza, strangles, or petechial fever, in which case we have so-called mixed infection.

**Course.**—The course of equine pleuro-pneumonia is ex-

ceedingly irregular, and may be typical, atypical, complicated, acute, chronic, or, not uncommonly, abortive.

1. A *typical course* is seen in horses which have a strong constitution, and which are affected either with the lobar form of pneumonia, or with a slight lobular affection of the lungs. The high temperature remains steady (*febris continua*) for from 5 to 8 days, after which it gradually falls, and the pneumonic or pleuritic symptoms disappear. Such cases usually end favourably; although the period of convalescence is longer (2 or 3 weeks) than that of influenza. Residence in a dry and well-ventilated stable, or in the open air, favourably influences the course of the disease. As soon as the first symptoms of the disease appear, the animal should at once be relieved from work, and should be carefully nursed.

2. *Abortive attacks* are usually met with during mild outbreaks and under favourable external conditions. The disease then lasts only for a few days and assumes the character merely of an infectious bronchial catarrh. These epizootic catarrhs of the respiratory mucous membranes generally precede the severer forms of pleuro-pneumonia. We find occasionally only premonitory symptoms of this disease. Sometimes, elevation of temperature is the only symptom present.

3. An *irregular course* with tendency to complications is generally found in weak animals; in those kept in unsanitary stables; in those which have been worked in spite of evident illness; and in horses suffering from gangrene of the lungs.

4. The *chronic course* of equine pleuro-pneumonia is attended by very serious consequences, and results from necrotic parts of the lungs becoming caseated and encapsuled, as in bovine pleuro-pneumonia. The contagium may remain virulent for months in these encapsuled foci. Generally, the only outward signs shown by the animal are loss of condition and inability to bear fatigue. Some horses become broken-winded, and others exhibit no marked sign of ill-health. Such chronic cases are a dangerous source of infection, if the encysted deposits communicate with a bronchus. The presence of such deposits may sometimes produce in the affected horse an acute and even fatal illness, after apparent recovery from the primary disease, as was shown in a case observed by Schwarznecker. In this instance, a 13-year-old cavalry horse contracted fatal inflammation of the lungs and pleuræ after a heavy field-day, 3 months subsequent to the disappearance of pleuro-pneumonia out of the affected squadron. This horse had remained healthy up to

that time, and his temperature, which was taken daily, was always found to be normal. *Post mortem* examination revealed in his lungs an old, encapsuled, caseous nodule, which, on account of the violent exertion, had burst into the thoracic cavity and had produced, there, an acute sero-fibrinous form of equine contagious pleuro-pneumonia.

**Diagnosis.**—The diagnosis of this disease is sometimes very difficult, on account of the not uncommon occurrence of abortive cases in which there are no pneumonic symptoms. Taking the temperature of the apparently healthy horses twice daily, is often the only means for the early recognition of the disease, when it breaks out among large numbers of horses which are kept together. This use of the clinical thermometer has proved of great service, especially in the army. When dealing with large numbers of horses, it is well, for prophylactic reasons, to regard as contagious pleuro-pneumonia, any feverish acute disease which shows lung symptoms, if it occurs without any assignable cause. We should, of course, exclude pneumonia due to chill, foreign bodies going the “wrong way,” metastasis, etc.

**Prognosis.**—The mortality of pleuro-pneumonia varies at different times. It depends on the progress of the epizootic, constitution of the animal, character of the nursing, amount of the contagium absorbed, and nature of the pneumonia. The course is generally favourable under well-regulated dietetic and hygienic conditions, and when the infected horse is well nursed. It has been observed, especially in the army, that the fact of withdrawing the affected horse at once from work and placing him in the open, has a good influence on the disease. The mortality, which varies from 20 per cent. to *nil*, is higher at the beginning of an epizootic than subsequently. During 1886 to 1893, over 17,000 horses suffering from pleuro-pneumonia, were treated in the Prussian army, with a loss of about 4 per cent. The proportion was the same in Bavaria. According to the observations of Dieckerhoff, the loss from this disease amounts to 15 per cent. In any case, the prognosis of pleuro-pneumonia is on the whole not so hopeful as that of influenza. The fact that the percentage of deaths is almost equalled by the percentage of cases in which pleuro-pneumonia leaves behind chronic disturbances of breathing after apparent recovery, makes the prognosis still more unfavourable. In connection with the necrotic disintegration of the lung tissue, we often find, in addi-



tion to the encapsuled necrotic deposits, the development of chronic intestinal inflammation, which leads to thickening and induration of the parenchyma of the lungs, with consequent diminution of the respiratory surface of the lungs. Chronic inflammation of the mucous membrane of the bronchi, with formation of bronchiectasis and peribronchitic deposits, are also met with. The exudative pleuritis not uncommonly gives rise to adhesions of the lungs with the thoracic wall or diaphragm, and in other cases to chronic hydrothorax and empyema. All these conditions cause chronic dyspnoea and decreased capacity for work, and thus form a not uncommon cause of "broken wind." To this must be added the other complications of contagious pleuro-pneumonia, such as roaring, inflammation of the tendons, paralysis, etc., which lead to chronic unsoundness.

**Therapeutics.**—The antipyretic treatment of pleuro-pneumonia, similar to that of influenza and of all other infective diseases, has not answered expectations; for its action has been found to be neither permanent nor very effective. For reducing temperature, we have generally to content ourselves with cold packing and enemata of cold water. Antipyretic remedies are necessary only when the elevation of temperature is very great, and long continued, or when there is a dangerous cardiac weakness. In such a case, we recommend antifebrin (doses of 30 grammes every 3 hours), digitalis, alcohol, and camphor. In a great number of cases we have used digitalis with very good results, but only in large doses. We give on an average 10 or 12 grammes of the leaves in a single dose. Instead of digitalis we may give tinctura strophanthi, 10 to 25 grammes internally, but not subcutaneously. We may give with advantage alcohol, the best form of which is wine (1 to 2 litres *pro dosi*); and camphor subcutaneously as spirits of camphor, or oil of camphor, 10 to 25 grammes each. The following are useful cardiac stimulants: caffeine (5 grammes *caffeinum natrio-salicylicum* dissolved in 10 grammes of water, subcutaneously), hyoscine (0.01 gramme), and atropine (0.05 to 0.1 gramme).

Priessnitz bandages and other counter-irritants are commonly used, and generally with good results in inflammation of the organs of the chest. We may also stimulate the activity of the skin by frictions with oil of turpentine (1 to 10 of spirits of camphor). One of the best blisters is oil of mustard diluted with alcohol (1 to 12-20). It has been strongly recommended by Dieckerhoff, and may be applied several times in case of need.

Cantharides ointment and croton oil should not be used. For the removal of the inflammatory products from the lungs, we may give expectorants, such as ammonium chloride, alkalies, and compounds of antimony; and to promote the absorption of the pleuritic exudate, we may employ diuretics and absorbent remedies, such as digitalis, alkalies, fructus juniperi, potass. acetat, and pilocarpin in doses of 0.1 to 0.2 gramme, or arecoline\* (0.08 gramme). Paracentesis thoracis is indicated if the amount of the pleuritic exudate causes danger to life. This operation, however, produces by no means such good results in pleuro-pneumonia as in rheumatic pleuritis; because the exciters of the inflammation continue to pass for a long time from the lungs into the cavity of the pleuræ. The severe intestinal affections are treated with small doses of calomel (1 to 4 grammes), or with neutral salts (Karlsbad salt). We may combat the formation of cavities in the lungs with inhalations of creolin, carbolic acid, and oil of turpentine, and internally with tar or oil of turpentine. If roaring should occur, small doses of strychnine (0.05 gramme subcutaneously), or of arsenic (liquor arsenicalis daily, 10 to 20 grammes) may be tried.

*Prophylaxis* is of the utmost importance. All the infected and suspected horses should be segregated without delay. The stables in which these animals stood should be thoroughly disinfected, if possible with solutions of corrosive sublimate, creolin, lysol, or with the strong fumes of chlorine. As soon as the infected horses have been taken away, their litter should be removed. The stables (hovels for preference) in which the infected horses are placed, should be well ventilated. The manure should be removed daily, and the floor daily disinfected with a 1 to 1,000 solution of corrosive sublimate, or a 3 per cent. solution of creolin. Care should be taken that the subsoil of the stable is in a sanitary condition. The temperature of the stable should be kept low in summer. The affected horses should remain in the open air as much as possible, and should be relieved from all work. Special attention should be paid to the convalescent animals; because they may continue capable of transmitting

\* Arecoline (an extract of areca nut) appears to possess, to some extent, the respective medicinal properties of eserine and pilocarpine. It strongly stimulates the glands, as shown by increased excretion of saliva and urine, and the peristaltic movements of the intestines, as made manifest by speedy defecation. It is recommended by Fröhner in the treatment of laminitis; and by Cadiot, in that of colic. The hydrobromate and the hydrochlorate are the chief salts used, in doses of from 0.05 to 0.1 gramme subcutaneously.—Tr.

the disease for a long time after they have apparently recovered, especially when necrotic deposits which communicate with a bronchus, remain in the lungs. Hence, convalescent horses should be kept under observation, and should be isolated for at least 6 weeks, until they are perfectly free from any suspicious symptom. It is also advisable to isolate all the horses of an infected stable which suffer from catarrhal or internal diseases. It is most important to disinfect the attendant veterinary surgeons and grooms, and especially their boots, clothes, and hands, and also the thermometers. New horses should be kept for 6 weeks under observation in quarantine. Opinions are divided as to the advisability, during an epizootic, of purposely allowing healthy horses to contract the disease. Experience leads us to believe that permanent immunity is not obtained by inoculations of the serum of the blood of horses which have passed through the disease. Consequently, this method of protective inoculation, which has been practised during the last few years, appears to be of no use.

**Skalma.**—Under this name Dieckerhoff describes several forms of equine disease, the chief symptoms of which are an irritable cough; nasal discharge and distressed breathing without percussion or auscultation revealing any morbid change; high temperature; loss of strength; and anæmia of the mucous membranes. The appetite remains comparatively good, and there is a rather long period of convalescence, which lasts usually 3 or 4 weeks. According to this, the disease may be regarded, on the whole, as an epizootic bronchitis which has a subacute course. It is supposed to be sometimes complicated with inflammatory irritation of the pharyngeal mucous membrane; and in severe cases with a diffuse exudative inflammation of the pleuræ (!). The course of the disease is generally mild. The disease appears in the form of a stable enzootic which may remain 2 or 3 months, or even longer, in a stable, and is said to be directly infectious. Dieckerhoff believes that "skalma" is one of the diseases which, like equine contagious pleuro-pneumonia and "equine distemper," were formerly included under the heading of "influenza."

We cannot acknowledge that skalma is a clinical unit. Some of Dieckerhoff's cases of skalma may have been closely related to pernicious anæmia. Its epizootic character, the well marked anæmia of the mucous membranes, inability to bear fatigue, irregularity in the rise and fall of the internal temperature in spite of the appetite remaining comparatively good, absence of any localisation with the exception of irritation of the respiratory mucous membrane, and long period of convalescence, also belong, according to our experience, to pernicious anæmia. On the other hand, we have seen cases of pharynx-angina and contagious pleuro-pneumonia which closely resembled those of skalma as described by Dieckerhoff. It seems that enzootic catarrh of the larynx and trachea have been described by some as skalma.

## TUBERCULOSIS.

General remarks on tuberculosis—Bovine tuberculosis—Tuberculosis of pigs—Equine tuberculosis—Canine tuberculosis—Tuberculosis of the cat—Tuberculosis of sheep—Tuberculosis of goats—Tuberculosis in wild mammalia—Tuberculosis of birds—Tuberculosis of parrots—Pseudo-tuberculosis.

**The Tubercle Bacillus and Tubercle.**—Tuberculosis is an infective disease that is caused exclusively by the bacillus tuberculosis, which was discovered by Koch in the year 1882. This bacillus is a slender rod of an average length of from 2 to 5  $\mu$  (about two-thirds of the diameter of a red blood corpuscle), and has rounded ends. It is easily stained with aniline dyes, and can be cultivated at the temperature of the body, on gelatine obtained from the blood serum of cattle. In 10 days these cultures form minute whitish streaks, and points which attain the size of a poppy seed in 3 or 4 weeks. The bacilli increase by transverse division, and also form small oval resisting spores \* which grow out into rods. Pure cultures inoculated in animals produce typical tuberculosis. The bacilli are chiefly found in the tubercles between and in the interior of the cells.

Tuberculosis is anatomically characterised by the formation of nodular, cellular, non-vascular foci (tubercles). The development of tubercle according to recent investigations by Baumgarten is effected by the immigration of the bacilli into the tissues with consequent change in the nuclei (karyokinetic nuclear division), and proliferation of the fixed tissue cells, which lead to the formation of a great number of epithelioid cells that have one or two nuclei. These epithelioid cells form nodular masses, namely, tubercles (large-celled tubercle). The single cells are separated from each other by fine connective tissue fibres (the reticulum of the tubercle). Sometimes the epithelioid cells attain a great size, and then contain several nuclei (giant cells). Simultaneously with this cell proliferation, an emigration of leucocytes takes place around the tubercle. If the number exceeds that of the epithelioid cells, the tubercle is called small-celled or lymphoid.

The tubercle, when it has attained its full development,

\* The chief characteristic of these spores, which are in their resting stage (endospores), is their great power of resisting external influences.

With our present appliances there is much difficulty in the microscopical identification of the spores of the tubercle bacillus.—T.R.

appears as a grey, translucent, hard nodule which is about the size of a millet grain (miliary \* tubercle), and which cannot be shelled out from the surrounding tissue. As its cells perish, it gradually undergoes retrograde metamorphoses (caseation or calcification). By the microscopical examination of a fresh typical tubercle we can, on making a section, easily distinguish three zones: 1. A central, round or angular, flat or spherical giant cell, with or without offshoots. It is granulated, and contains from 20 to 30 circumferentially-situated nuclei, and sometimes numerous tubercle bacilli. 2. A zone of large epithelioid cells which have round or oval nuclei, and which contain 1 or 2 bacilli. 3. An outer zone with numerous small, round cells that have strong nuclei (granulation tissue) between which a fine reticular connective tissue lies. The giant cells and the epithelioid cells are sometimes absent.

**Biology of the Tubercle Bacillus.**—The well-marked power of resistance to various agents possessed by the virus of tuberculosis is due to the fact that the bacilli form resting spores. The tubercle bacilli perish very quickly outside the animal body in temperate climates; even sunlight of itself is able to kill them in periods varying from a few minutes to a few hours. Numerical increase or further development of the bacillus outside the animal or human body in the manner of a miasma is consequently impossible. Tuberculosis is therefore to be looked upon as a purely contagious disease. The tubercle bacilli are not ubiquitous (Cornet), and are comparatively resistant. They remain, for instance, virulent in ordinary water for 120 days (Cadéac); in the water of the Seine for 50 days according to Chantemesse, at 8° to 12° C.; and for 17 days at 15° to 18° C. Tuberculous sputum of man continued capable of infection, on one occasion, for 179 days; and on another, for 226 days. When in a dried condition it retains its infectiousness for some years (Strue). With respect to the influence of dilution on the

\* S. J. Gee and Percy Kidd (Quain's *Dictionary of Medicine*) remark: "Miliary tubercle may or may not be of the size of a millet seed. Tubercle the size of a millet seed may or may not be miliary. All that is now meant by miliary tubercle is a small nodule, roundish, seldom larger than a hemp-seed, almost colourless or greyish, consistence almost equal to that of cartilage, and either quite transparent or opalescent. Lastly, miliary nodules, may or may not be tubercular."

The miliary tubercle, which is the primary lesion produced by the bacillus tuberculosis, is non-vascular; a fact which appears to be closely connected with the first degenerative change undergone by the grey granulation, namely, caseation. The subsequent retrogressive changes of calcification and fibrosis are methods of healing.

The use of the term "tubercle" (*tuberculum*, a small swelling) has, since the discovery of the tubercle bacillus, become more and more restricted to changes connected with tuberculosis. The expression "nodule" or "tumour" now, as a rule, replaces "tubercle" in the description of other diseases.—T.R.

action of tubercle bacilli, the investigations of Bollinger and Gebhardt have shown that the bacilli even after having been diluted to 1 to 400,000 produce tuberculosis when administered subcutaneously or intra-peritoneally. The fact of diluting the virus seems to proportionately decrease its power of infection only when it is given by the mouth, as in food. Dry sputum becomes sterilised in less than 15 minutes when it is exposed to steam at 100° C., and by dry heat in one hour (Schill and Fischer). Bang states that raising tubercular milk to a temperature of 85° C. at once kills the tubercle bacilli in it. These microbes, according to Forster and De Man, are destroyed by a temperature of 55° C. continued for 4 hours; 60° C., for one hour; 65° C., for 15 minutes; 80° C., for 5 minutes; and 95°, for one minute. A solution of from  $\frac{1}{10}$ th to  $\frac{1}{100}$ th per cent. of corrosive sublimate in quantities of from 8 to 12 times those of the dry sputum of tuberculous people, will render it sterile in from 20 to 24 hours. The same effect is produced by a 5 per cent. solution of iodide of potassium, iodoform in powder or in vapour, iodine vapour, 1 per cent. solution of caustic soda, and 5 per cent. watery solution of creolin or carbolic acid. On the other hand, a saturated solution of common salt, water containing 1 per cent. of bromine, or  $\frac{1}{10}$ th per cent. of iodine, iodoform dissolved in oil or turpentine (Schill and Fischer), alcohol, putrefaction, desiccation, freezing as low as -8° C. (Galtier), and a solution of soap (Guinard), are ineffectual for this purpose.

**General Remarks on the Pathogenesis of Tuberculosis.**—As the immigration and the dissemination of the tubercle bacilli in the body are not uniform in all species of animals, and as cattle, pigs, and poultry, in particular, exhibit a special mode of invasion and distribution; we shall deal here only with the general principles of the pathogenesis of tuberculosis, and shall discuss special points and peculiarities more minutely when we refer to the respective species.

The tubercle bacilli enter the body most frequently along with the respired air\* and through the alimentary canal. Consequently the lungs and the intestines are the chief ports of entrance for the virus. At first, tuberculosis always appears as a local affection which usually becomes developed at the place of entrance of the bacilli (lungs or intestines). However, it not unfrequently occurs that the organs through which the bacilli entered, especially the lungs and intestines, remain free from the disease, and that the tubercular process becomes

\* Klein (*Micro-organisms and Disease*) states: "I have had guinea-pigs kept in their cages in the ventilating shaft of the Brompton Hospital [for consumptives], and have thereby produced general tuberculosis in the great majority of them: caseous tubercles in the lungs, lymph glands, spleen, liver, pelvic glands were the result; thus proving that the air of any place where tuberculous persons sojourn, contains the tubercle virus, and must therefore be considered as not free from danger."—TR.

localised, first in the bronchial, or mesenteric glands, and from thence in the pleuræ or peritoneum, after the tubercle bacilli have passed through the respiratory or digestive mucous membranes without injuring them. The local affection consists in the formation of miliary tubercles, which, when in considerable numbers, may become combined together, so as to make a granulation tumour. These infectious new-growths can exist for a considerable time as localised foci of inflammation, or they may become calcareous, caseated, or undergo fibrinous degeneration. Or they may ulcerate, suppurate, or form cavities by disintegration. The disease may become cured by entire calcification of the tubercles, or by the formation of connective tissue completely separating them from the healthy parts.

From these local foci (localised tuberculosis), the tubercular process can, as is most frequently the case, spread in the following ways, particularly in consequence of the breaking down of the tubercle. 1. By the *lymphatics*. Secondary tubercles spring up, first of all in the neighbourhood of the primary tubercle. After this, the lymph glands especially become infected; in pulmonary tuberculosis, the bronchial glands and the mediastinal glands; in intestinal tuberculosis, the mesenteric glands, spleen, and liver; in tuberculosis of the upper air passages, tracheal glands, upper, middle and lower cervical glands; etc. This affection of the lymph glands is always present in tuberculosis. The extension of the disease to the lymphatics very frequently goes on from the freshly inflamed lymph glands, to serous membranes, particularly those of the pleuræ and peritoneum. Furthermore, tuberculosis of the abdominal cavity, without the agency of the blood vessels, may spread to the thoracic cavity, by the bacilli passing through the finely fissured interstices of the diaphragm. 2. By *continuity*, especially by means of the *mucous membranes*. For instance, tuberculosis of the mucous membrane of the bronchi and larynx may, by the breaking into them of the neighbouring tubercular deposit, be at first set up, and later on tuberculosis of the intestine, from the swallowing of bronchial secretions which contain the bacilli. In a similar way, tuberculosis of the genito-urinary organs may become developed from the presence of the disease in the kidneys or genital glands. 3. Extension of tuberculosis by means of the *blood-vessels* may take place, when one of the large veins, on becoming opened by erosion due to the breaking down of a tubercular focus (perhaps in the lungs), takes up part of the disintegrated mass and thus introduces it into the blood

current; when a tubercular affection forms in the wall of a blood-vessel, as a consequence of the extension of the disease from a neighbouring focus; or when the bacteria are transmitted along with the lymph into the thoracic duct. In these cases, the bacilli may be carried by the blood into distant organs, and thus produce general tuberculosis which may be divided into two principal varieties, namely, chronic general tuberculosis, and acute, miliary tuberculosis. To *chronic general tuberculosis* we refer those cases in which, as a consequence of an injurious immigration of bacilli into the blood, only isolated tubercular foci form in various organs. These deposits at first are small; but subsequently they gradually enlarge by aggregation into nodules and tubercles. On the other hand, generalised tuberculosis is called acute miliary tuberculosis, when a determination of blood rich in bacteria takes place. This *acute miliary tuberculosis* is characterised by the simultaneous invasion, in all organs, of innumerable minute (miliary) tubercles. The lungs and the liver are specially liable to tubercle on account of their comparative vascularity. Chronic general tuberculosis is not infrequently complicated with acute miliary tuberculosis.

The somewhat rare cases of foetal (placental) tuberculosis can be explained, partly by infection of the foetus through the mother, the bacilli having penetrated the placental membranes, as has been experimentally demonstrated with animals (guinea-pigs and rabbits), and partly by a primary tubercular affection of the ovum at the time of coition. A germinal tuberculosis, namely, one inherited from the father, is extremely rare. The bacilli of this disease are very seldom found in the semen of tuberculous male guinea-pigs whose sexual organs are not the seat of tuberculosis. The number of tubercle bacilli in the semen of guinea-pigs which suffer from tuberculosis of the testicles, though somewhat greater, is still very small. When such animals were paired with healthy females, the offspring was found to be thoroughly sound and free from tuberculosis (Gärtner). The widely spread assumption that male animals kept for stud purposes are an important factor in the dissemination of tuberculosis, by their transmitting the disease in the act of copulation, is therefore untenable. Hereditary predisposition to tuberculosis is a subject upon which scientific research has thrown no light.

**Tuberculosis transmitted by Food.**—The idea that



tuberculosis is a specific infective disease is only of recent date ; for the researches on this point spread over a period of rather less than thirty years. Prior to that time, tuberculosis was looked upon neither as an independent pathological unit, nor as an infective disease. It was only by numerous experiments in feeding and inoculation that the infective and uniform character of tuberculosis was clearly demonstrated. The first inoculation experiments were made by Villemin in 1865, from which time began the etiological investigation of this disease. Villemin transferred, partly subcutaneously, partly intra-tracheally, human tubercular matter to animals, and was the first to establish the fact that tuberculosis is a specific infective disease, the cause of which is an inoculable agent. He further asserted that tuberculosis of man was identical with the "grapes" or consumption of cattle. Klebs inoculated the serous cavities of the body and made the first experiments with food. By these means, he established the identity of tuberculosis of cattle with that of man, and was the first to lay stress on the danger of the milk of tuberculous cows being given to children. Chauveau (1868) also demonstrated, by numerous convincing experiments, the transmissibility of tuberculosis, by means of food, and declared that the consumption of the flesh of tuberculous animals was hazardous. The results of his researches were confirmed by Villemin, St. Cyr, and other French investigators.

Gerlach, along with Klebs, at the Hanover veterinary college in 1868, was the first in Germany to make exhaustive feeding experiments. After feeding with tubercles and with the milk of tuberculous cows, he succeeded in 7 experimental animals out of 8, in producing tuberculosis of the lymph glands, lungs, intestines, and liver. Gerlach had previously declared that the milk and flesh of tuberculous animals were infectious. He obtained, later on, similar results with 46 feeding experiments at the Berlin veterinary college. During the years 1870 to 1873, Günther and Harms undertook, in Hanover, 94 feeding experiments with animals of widely differing species and with various kinds of tubercular material, and obtained 24 positive results and 70 negative or doubtful ones. The majority of the 14 experiments made in 1870 and 1871 at the Dresden veterinary college gave positive results. The same may be said of the experiments of Zürn, Bollinger, and Roloff.

As these positive results were discounted by many negative

ones; the theory of the transmissibility of tuberculosis by flesh and milk from animals to man, met with, at first, severe opposition and denial. Thus, in 1876 the German Veterinary Council rejected Gerlach's assumption. During the following year, positive results were produced by Blumberg, Lange, Orth, Toussaint, and Peuch, and by experiments at the Berlin veterinary college during the years 1876 to 1880, and at the Dresden veterinary college in 1878 and 1879. Besides, many cases of accidental transmission of tuberculosis through milk, sputum, and flesh, to men and animals were proved.

The most important year in the history of research in tuberculosis was 1882, in which Koch discovered the tubercle bacillus. From thenceforth the possibility of transmission was no longer doubtful. Thus, Baumgarten produced tuberculosis in every case by feeding with milk which had been mixed artificially with cultures of bacilli, and Haug invariably obtained positive results by experimental feeding with milk taken from tubercular udders of cows.

Wesener compiled reports, up to 1884, of 369 feeding experiments, the positive and negative results of which were about equal in number. We find from this compilation that—(1) 71 animals among which guinea-pigs and swine proved most susceptible, were experimented upon with human tubercular matter. (2) 180 experiments were made with tubercular matter from cattle and other animals. More than three-fourths of the cases of inoculation were successful in calves, goats and sheep; three-fourths, in pigs; one-half, in rabbits; over one-third, in cats, and somewhat less than one-third, in dogs. (3) The flesh of tuberculous cattle was given on 32 occasions as food, with the result that pigs were found to be more susceptible than other animals, and that dogs were unaffected. Boiled flesh produced no effect. (4) The milk of tuberculous cows was given as food in 86 cases, out of which, three-fourths of the pigs and half of the goats became diseased. Boiling infected milk decreased its virulence. In the scale of comparative racial susceptibility, the herbivora (cattle, sheep, and goats) proved highest; then swine; and after them, guinea-pigs and rabbits. Carnivorous animals (cats and dogs) were very little affected, and birds least of all with the exception of domestic fowl. The tubercular matter of animals was found to possess the greatest power of infection; then came the sputum of tuberculous men and the milk of tuberculous animals; and, lastly, tuberculous flesh.

Various circumstances have contributed to render unsuccessful the numerous feeding experiments that have been made. In the first place, the material given as food frequently contained no tubercle bacilli, or extremely few. These bacteria, as is well known, are absent in caseous deposits and in calcareous

bovine tubercles ; in fact, bovine tubercles are, on the whole, poor in bacilli. The flesh also is seldom the seat of tuberculosis, and the milk, as a rule, only when the udder is affected. The virulence of experimental food was frequently weakened, or completely destroyed by putrefaction. The shortness of the time during which the bacilli remain in the stomach and intestines frequently prevented infection. In many cases, the experimental animals were more or less unsuitable. Finally, it appears that the dilution of the material containing the introduced bacilli by the other fodder, and especially by the drink and the digestive fluids, lessened the activity of the bacilli, which result might also have been promoted by the direct influence of the digestive fluids. To clear up this point, Wesener made a series of feeding experiments by the direct introduction, into the stomach and also into the intestines of a rabbit, of human tubercular sputum, respectively fresh, dry, putrescent, and after it had been subjected to the action of the various digestive juices. The investigations of Strauss and Würtz have also shown that the gastric juice weakens the virulence of the bacilli, and entirely destroys it in 6 hours. We may infer from these and other researches, that the natural gastric juice, if allowed to act for a sufficiently long time, weakens the vitality of the bacilli, and even kills them ; but it certainly does not produce this effect on the spores, which may get into the intestine, and, without affecting the intestinal mucous membrane, may set up tuberculosis of the mesenteric glands (*tabes mesenterica*). In this respect, milk\* which contains both bacilli and spores, is the most dangerous agent. Flesh is much less so ; because, as a rule, it is free from bacilli, or contains them only in small numbers, and is usually consumed in a cooked state. Experimental inoculations which Bollinger made in guinea-pigs with the juice expressed from the flesh of tuberculous cattle, gave negative results. The experiments of Bang, Zschokke, Bollinger and others have shown that milk may contain bacilli or spores without the udder being affected, and may therefore act as an infective agent. Recent experiments of Gebhardt have made it probable that the virulent milk of tuberculous cows may be rendered innocuous by considerable dilution (1 in from 50 to 100), by, for instance, the admixture of the milk of healthy cows.

\* Klein suggests that the frequency of miliary tuberculosis in children is due to the ingestion of tuberculous milk. Sims Woodhead refers the high percentage of *tabes mesenterica* among children to the same cause.

**Occurrence.**—Tuberculosis, leaving man out of the question, is most frequently observed in cattle, pigs, and birds, and less often in carnivorous animals and horses: The apparent rarity of tuberculosis in goats and sheep is due to the absence of special research on this subject. Tuberculosis occurs very frequently in apes, monkeys, lions, tigers, bears, and other wild animals (dromedaries, giraffes, jackals, panthers, jaguars, and arctic foxes) which are kept in captivity; in rodents; and apparently in amphibia (Lechner). Field mice and white mice are immune. In Japan, the indigenous breed of cattle, contrary to what is the case in imported and half-bred cattle, are free from tuberculosis (Janson).

[Sidney Martin states that one-seventh of mankind dies of tuberculosis; and that the bacillus is a parasite, and not a saprophytic micro-organism, although it may be cultivated in specially prepared media outside the animal body. Kanthack (Allbutt's *System of Medicine*) on the contrary, considers that it is nearly certain that this microbe is capable of a saprophytic existence; that healthy persons are relatively immune; and that improved hygiene and the destruction of the sputum of consumptives, are the best means for checking the spread of the disease among mankind. He argues that conclusions drawn from experiments with guinea-pigs should be received with great caution as regards their application to man; because these rodents are extremely susceptible to tuberculosis, "and that the healthiest specimens, if inoculated with the smallest quantity of mammalian tubercle bacilli, will succumb."—Tr.]

#### BOVINE TUBERCULOSIS.

**History of Tuberculosis in Cattle.**—Tuberculosis of cattle is one of the oldest known diseases of our domestic animals. The Mosaic laws (Leviticus xxii. 22) contain rules that the flesh of animals which suffer from "wen or scurvy" should not be used as food. The Talmud, especially the Mischnal (third century), and the Gemara (fifth century), contain numerous enactments against the eating of such flesh. The "kandi" and "timari" in these books probably mean tubercle. It is evident that in the eleventh and twelfth centuries, tuberculosis was fairly well known to the Arabian Rabbi Isaak Alfasi, the Hebrew physician, Maimonides, and the French physician Raschi (1105). The book, *Schulchan Aruch*, which is a Jewish collection of rules on this subject, has been handed down to us from the sixteenth century.

The Franks had in Germany, in the ninth century, ecclesiastical laws against the eating of the flesh of cattle and pigs

which were affected with tuberculosis of the serous membranes (*kadrerie*). In the year 1370 it was forbidden in Munich to have on sale the flesh of tuberculous animals. Similar laws were passed in 1343 in Würzburg; in 1394, in Passau; in 1401, in Landshut; in 1558, in Württemberg; in 1582, in the Palatinate; etc. It is reported that 12 students who had eaten the flesh of tuberculous animals, died in Leipzig in 1677. In 1702 Florinus gave a description of the symptoms of the disease during life. Even at that time the name of "French disease" was in common use. The name arose from the fact that tuberculosis was considered to be connected with human syphilis, which was regarded as its cause (bestiality). The term *Franzosenkrankheit* is said to have been first used by Helmont. In consequence of this theory of origin, all tuberculous cattle had to be destroyed. Thus, for instance, the yearly loss arising from the slaughter of affected cattle in the Prussian countries amounted, according to Graumann (1784), to about £900. Severe penalties were enacted against the violation of the sanitary laws.

In 1783, the Berlin Board of Health first published inspection of meat regulations, in which it described the characteristics of "French disease;" rejected the theory of its connection with syphilis, and declared the flesh to be fit for human food. In the same year, Kersting of Hanover expressed a similar view in a report to the government of Mecklenburg-Strelitz. Franck of Baden was of opinion that the private consumption of the flesh of cattle which were only slightly affected with tuberculosis, might be permitted. Graumann declared in 1784, in a government order of Mecklenburg-Schwerin, that such meat might be eaten. In consequence of this, all the orders which had been made against the consumption of the flesh of tuberculous cattle were cancelled throughout Prussia in 1785; and throughout Austria in 1788. Tscheulin, in 1816, laid down with regard to the inspection of meat, three degrees of bovine tuberculosis: (1) in which only the tubercles were to be removed; (2) in which the diseased parts were to be destroyed and the meat to be sold at a cheap rate; and (3) in which the flesh was declared to be entirely unfit for food. A similar procedure, as regards meat inspection, was carried out, up to a very recent date, in Southern Germany, Switzerland, Austria, France, Belgium, Spain, Italy, and other countries.

Phthisis and bovine tuberculosis were for a long time considered to be two different diseases. Thus Virchow, Schüppel,

and others declared that the tubercles in cattle were lymphosarcomata. Leisering regarded them as sarcomata. On the other hand, Spinola and Haubner maintained that bovine tuberculosis and human tuberculosis were identical. This identity has, however, only during the last decade, been positively proved by the feeding experiments of Gerlach and the bacteriological results of Koch.

**Etiology.**—The penetration of the tubercle bacillus into the tissues of the body is undoubtedly the sole cause of the disease, although the mode of infection may vary. Usually the infection is manifestly transmitted by cohabitation. Thus, many cases are known in which a new-comer infected in turn, all the other residents of the cow-shed. In such instances the infection is mostly brought about by coughed-up tubercular matter, which dries and becomes mixed, in the form of dust, with the inspired air. On the other hand, the researches of Cornet, Cadéac, Malet, Celli and Guarineri, show that we must not assume that the tubercle bacilli are exhaled into the air by the affected animals and are then inhaled in a pure condition by their neighbours. In a smaller number of cases, as Bang has shown, the transmission of the bacilli might take place, *per os*, by the animals licking each other and by the consumption of sputum and bacilli which have been deposited from the air, in the form of dust, on the fodder. This theory receives support from the frequency of the affection in the upper cervical glands, and from the fact that in a cow-shed which has a common fodder trough,\* the tuberculous animals which stand above the others infect them, and that the spread of the disease can be stopped by simply changing the position of the cattle. The disease is very often transmitted to calves by the milk of tuberculous cows, especially when the udder is affected. The milk of tuberculous cows may, as we have already said, contain bacilli and spores without the udder being diseased. Investigations of Heim, Leser, Bang, Roth and others have also shown that dairy produce (whey, cheese, and butter, for instance) from tuberculous cows may transmit the disease, even after a lapse of from one to four weeks. Bang has proved that although the centrifugal process deprives tubercular milk of a large amount of its poison, by hurling the greater number of the bacilli into the sediment, it does not afford absolute

\* The trough here alluded to has a downward slope from one end.

protection from infection; because skimmed milk and cream prepared by centrifugal methods have, on inoculation, been found capable of setting up tuberculosis. The existence of congenital (fœtal) tuberculosis has been amply proved by a large number of observations. Here we must distinguish between a placental infection, namely, the infection of the embryo through the diseased mother, and a conceptional or germinative infection, namely, the infection of the ovum, through the semen, during coition. The possibility of placental infection has been proved by frequent veterinary observations. Its occurrence depends on a previously diseased condition of the placenta as Sanchez has shown experimentally with pregnant rabbits, which, by inoculation, contracted the disease without the fœtus becoming affected, as long as the placenta remained intact. The far rarer germinal affection has been shown to be possible by the experiments of Marfucci, who inoculated hens' eggs with cultures of the bacilli and obtained well-marked tuberculosis in the chickens. The not unrare cases of tuberculous infection during the first months of fœtal life, generally terminate in early abortion; although the disease has on several occasions been found in newly-born calves. Infection through coition from the bull to the cow, and *vice versâ*, is less frequent; though the possibility of its occurrence has been proved by many practical observations. On the other hand, it is still an open question whether cattle have ever been infected by tuberculous people.\* Certain cases of intestinal tuberculosis have been mentioned which were stated to have been caused by feeding on swill and kitchen refuse. Such a possibility cannot be doubted if we take into consideration the many well-proved cases of

\* Dr. Lingard, who is the Imperial Bacteriologist of India, tells us in his *Annual Report* for the year 1902-1903, that "the results of Professor Koch's experiments in Europe showed that bovines inoculated with the *materies morbi* of human tuberculosis failed to bring about any pathological changes in the inoculated animals." In order to test these experiments, he inoculated intravenously a 3 year old bull with a cultivation of tubercle bacilli derived from human sputum grown in glycerine bouillon; and a 1 year old bull, with fresh sputum containing a large number of tubercle bacilli obtained from a case of human tuberculosis of the lungs. In both cases, the results were negative. "Control experiments were at the same time made in rabbits with the result that these animals succumbed in an average of 92 days after inoculation." He therefore draws the conclusion that "the bacillus of tubercle obtained from a human source is unable to reproduce the disease in bovines. There is therefore an indication that tuberculosis in human beings might be retarded, if not cured, by the continued administration, during a long period, of healthy bovine serum, either subcutaneously, or *per os*."—Tr.

tuberculosis having been transmitted from men to other animals, such as fowl. Lydtin reports a case of accidental transmission of tuberculosis by an inoculation which was intended to obtain protection against pleuro-pneumonia contagiosa, and which was made with material taken from a beast suffering from both pleuro-pneumonia contagiosa and tuberculosis. Bang suspects that a primary affection of the udder may take place in consequence of the bacilli entering externally through the teats.

The bacilli may penetrate into the body through intact mucous membranes. This entrance is facilitated by certain predisposing influences, which were formerly considered to be the real cause of tuberculosis, such as feeding on innutritious and watery food, and prolonged residence in badly ventilated and crowded sheds. The fact that depastured cattle, especially on mountains, become much less frequently affected with tuberculosis than stall-fed cattle, is the best proof that the vitality of the respiratory organs becomes weakened in cattle which are kept in sheds and which consequently become predisposed to the penetration and deposition of tubercle bacilli in their bodies. The penetration of the bacilli is greatly facilitated by the presence of catarrhal affections of the respiratory mucous membrane, which are caused or favoured by the state of the air in the shed, especially when there is an accumulation of secretion in the bronchi. The disease caused by arsenical fumes is, according to Johne, a true chronic inhalation-tuberculosis, arising from the inhalation of these fumes first of all setting up an inflammatory affection of the mucous membrane, which facilitates the invasion of the bacilli. The predisposition to tuberculosis is also increased by an abundant production of milk and numerous births, both of which weaken the system and impair its power to resist the penetration of the bacilli. For similar reasons, the disease frequently becomes much worse directly after a birth, or becomes well-marked when puberty is attained. Continuous in-breeding and attempts at establishing pedigree stock are predisposing factors which account for the susceptibility to tuberculosis of animals bred from closely related parents. An inherited predisposition seems not uncommonly to occur by reason of the constitutional debility of the mother being transmitted to the offspring.

**Occurrence.**—Tuberculosis is by far the most widely-spread bovine disease. It is found in nearly all countries and regions of the world, and most frequently in the neighbourhood of



cities. In the polar regions, the North of Sweden and Norway, North Africa (Texier and Cochez), the steppes of Russia (Spinola), and in some isolated and but little frequented islands, as for instance, Iceland (Krabbe) and Sicily (Chicoli), the disease occurs very seldom or not at all. As we have already said, it does not appear among Japanese cattle (Janson) or wild cattle (Veith). The fact is accepted in Germany that in secluded districts and parishes in which close attention is paid to maintaining the purity of the local breeds, tuberculosis is rarer than in places where there is much traffic in cattle and where foreign cattle are imported. Bang made similar observations respecting certain Danish islands; and Krajewski, as regards the grey cattle of the South Russian steppes.

The *percentage of cattle suffering from tuberculosis* varies much in different districts. In Germany, according to the statistics collected in the year 1888-1889 by the Imperial Board of Health, from 2 to 8 per cent. of all the cattle were found to be affected with tuberculosis. These figures are, certainly, far too low; for the positive results with tuberculin inoculations have not infrequently been as high as 50 per cent. Besides, it is evident that tuberculosis is universally increasing among cattle. For instance, it was found in the Leipsic abattoir in the year 1890, that 22 per cent. of all the slaughtered animals were tuberculous; in 1892, 27 per cent., and in 1894, 29½ per cent. The percentages in the different states were: Prussia, 5 (about equal to 6 or 7 per cent. of all the cows); Saxony, 8; Bavaria, about 3; and Baden and Hesse, 2. The different provinces show the following percentages: Pomerania, 16; Magdeburg, 12; Lower Silesia, 11; Schleswig-Holstein, Upper Silesia, and Posen, 7 to 10; Hanover, Brandenburg, Bromberg, Breslau, Erfurt, and Merseburg, 3½ to 5; West and East Prussia, 3; Westphalia, 2; and the Rhenish Province, 1 to 2½. The percentages of cases met with in the abattoirs of these places are naturally much higher, and differ according to the district. Eight per cent. of all the slaughtered cattle were found to be tuberculous in Prussia in the year 1893-1894 (64,000 tuberculous among 777,000 slaughtered); in the districts of Magdeburg and Stralsund, 16; Coblenz, 12; Berlin and Lüneburg, 11; Marienwerder, Cöslin, Liegnitz, and Oppeln, 10; Merseburg, Hildesheim, Wiesbaden, and Aurich, 9; and Danzig, Frankfurt, and Stettin, 8. In the abattoirs of Saxony in 1889, the percentage of tuberculosis in all the cattle slaughtered, varied from 1 to 16; in the year 1888, from ½ to 22½, and in 1892,

1893, and 1894, from 18 to 21½. Of the cattle slaughtered, from 1888 to 1894 in Leipsic, more than 20 per cent. (15 to 27 per cent.) were affected with tuberculosis. The number of animals found to be tuberculous in the abattoirs in Munich and Augsburg, amounted to from 2 to 11 per cent.; in Berlin, 2 to 5; Chemnitz, 3 to 5; Lübeck, 8 to 9; Greifswald, 9 to 10; Kiel, 5; Goldberg, 20; Minden, 3; Frankfurt, 3 to 4; Duisburg, 4; St. Johann and Zittau, 8 to 9; Mittweida, 5 to 10; Wismar and Schwerin, 13 to 14; Lübeck, 2 to 3; Hagenau, 6; Dornach, 10 to 11; Metz, 3; Strassburg, ½ to 2; Baden, 1 to 2; Bremen, 2; Copenhagen, 16; Paris, 6; and in Belgium, 4. It was found in England during the year 1892, when cattle were being slaughtered on account of contagious pleuro-pneumonia, that from 20 to 30 per cent. were tuberculous. The percentage in London was from 25 to 40; and in Edinburgh, Yorkshire and Durham, 19 to 23.

Lowland *breeds*, such as Dutch cattle, are much more frequently affected than the mountain breeds, which possess stronger constitutions. Cattle kept in sheds, as we have already remarked, are far more susceptible than those on grass. With regard to sex, we find that cows and heifers are more liable to tuberculosis than bulls and oxen. We learn from the foregoing statistics that out of every 223 animals, 170 cows, but only 38 oxen, 14 bulls, and 1 calf suffered from tuberculosis. It is likely that this disease is most prevalent amongst the milch cows of dairies, distilleries, breweries, sugar refineries, etc. Old animals are much more frequently tuberculous than young ones. According to the before-mentioned statistics, the cases of bovine tuberculosis, as regards age, during 1888 to 1889 in the German Empire, were as follows: 43 per cent. were 6 years of age; 33 per cent. from 3 to 6 years; 1 per cent. under 1 year old; and 0.4 per cent. under 6 weeks. Among a million calves which were killed in Munich from 1878 to 1882, only 5 were found to be suffering from congenital tuberculosis. Among about 80,000 calves that were slaughtered in Berlin in 1885-1886, only 7, and amongst about 90,000 killed in 1886-1887, only 6 were tuberculous. Only 9 cases of tuberculosis were met with in 230,000 calves which were killed in Augsburg from 1873 to 1886; and 73 cases (0.02 per cent.) in 370,000 calves in Prussia during the year 1888 to 1889. The percentage of cases of tuberculosis in newly-born calves is reduced by the fact that tuberculosis is an exciting cause of early abortion.

**Anatomy.**—The anatomical changes in bovine tuberculosis are mostly found in the lungs (phthisis) and in the serous membranes of the large cavities of the body ("grape disease").\* In about half of all the cases, the lungs and the serous membranes became simultaneously affected; in about a third, the lungs alone; and in about one-fifth, the serous membranes only. When the lungs and serous membranes are attacked, their respective lymph glands always become affected at the same time. In constitutional general tuberculosis, all the other organs of the body may show tubercular changes, which, however, may be restricted even to one organ (compare the figures on page 172).

1. In the *lungs* we find, first of all, circumscribed caseous pneumonic foci of various sizes, which develop from a catarrhal pneumonia, with atelectasis (imperfect expansion) of the alveoli and bronchioles, emigration of leucocytes, and accumulation of large epithelioid cells in the interior of the alveoli (desquamative pneumonia), and subsequent caseation or suppuration of the affected parts of the lungs. This gives rise to the formation of caseous foci and cavities with yellowish, caseous, crumbling, and greasy or purulent contents. These changes are accompanied by chronic indurating inflammatory processes in the interstitial tissue of the lungs. These processes appear chiefly in the neighbourhood of the caseous foci and miliary tubercles; and manifest themselves by an interstitial new growth of connective tissue, and carnification and shrinking of the lung tissue, which sometimes shows an almost cartilaginous or flesh-like thickening and even complete calcification, and consequently offers a certain amount of resistance when it is being cut through.

Another kind of tuberculosis of the lungs manifests itself, in the presence of the miliary tubercles, as nodules, varying in size from a millet seed to a pin's head. They are pale-yellow, of moderately firm consistence, and are only slightly translucent. They are usually spread in great numbers, either over the surface of the lungs, or prominently on the surface of the section; and at first are surrounded by thoroughly healthy lung tissue. Later on, these nodules become caseated and calcareous in their centre. Large tubercular nodules are frequently formed by the accumulation of several of these miliary tubercles, which,

\* In Germany, the expression *Perlsucht* (pearl-disease) is used to signify tuberculosis of the pleuræ and peritoneum, and corresponds, more or less, to the English term, "grape disease." We may translate *Perlen* by "grapes."

in old standing cases, may undergo caseation or calcification in their interior.

The bronchial mucous membrane is often in a state of chronic catarrh in which greater or less bronchiectasis (dilatation of the bronchi) is not rare. Also, tubercular ulcers and miliary tubercles may occur in the bronchial mucous membrane and in the laryngeal mucous membrane; the ulcers being distinguished by thickened, turned-up edges, and the miliary tubercles by the fact of their being arranged in rows. The tubercles spread usually from the bronchial mucous membrane to the peribronchial connective tissue (bronchitis and peribronchitis nodosa) and also to the lung tissue.

The bronchial glands are always swollen, enlarged and infiltrated with miliary tubercles. Later on they become caseous and calcareous.

On the pleuræ there may become developed a circumscribed, chronic, adhesive pleuritis, which is followed by adhesion of the lungs to the thoracic wall. Sometimes the lungs show at the same time other specific changes, such as those caused by echinococci, pleuro-pneumonia, etc.

2. Tuberculosis of the *pleuræ* and *peritoneum* ("grape disease") begins with the formation of very minute, light grey, translucent nodules ("grapes") which are at first smaller than a grain of sand, and give rise to the granulated condition of the surface of the pleuræ and peritoneum. An abundant new-growth of connective tissue forms round these tubercles ("grapes") with greatly increased vascularity of the serosa, so that the tubercles are, as it were, imbedded in a frame of connective tissue.

On account of the confluence of several of such tubercles and the simultaneous proliferation of the connective tissue stratum, nodules, varying in size from a lentil up to a pea, now form partly on the visceral and partly on the parietal membrane, and may finally become as large as a hen's egg or as a man's fist. At first they are of a soft, juicy, gelatinous consistency, and of an orange colour, and, on a section being made, often show a dark-red centre. Later on they become harder and firmer, assume a fibrous or connective tissue-like consistency, and acquire an uneven nodular surface, so that they grate on being cut through. Or they undergo caseation or calcification in their centre, which then consists of a brittle, mortar-like, grey-yellow substance. In this more advanced state, the colour of the tubercles is light grey, ash-grey, or bluish white.

These tubercles ("grapes") assume very characteristic forms. By large numbers of them being variously grouped together, they may present a villous, wart-like form, or racemose appearance; or they may take the shape of a cauliflower or mulberry. Sometimes they have a broad base, at other times they are pedunculated or shaped like a pendulum or polypus. They may cover the entire surface of the serous membrane, and may become enormously increased in number. These tubercular new-growths frequently attain a weight of from 30 to 40 kilogrammes and even more. The individual nodules have, at this stage, either grown firmly together, or have become united by bands or threads of connective tissue.

The anterior and posterior *mediastinal glands* become enlarged in the same way as the bronchial glands. They become infiltrated with miliary tubercles, indurated or caseous, and changed into tumours of greater or less size, even up to half a yard long. The enlarged mediastinal glands frequently enclose and compress the œsophagus.

3. Besides the bronchial and mediastinal glands, a considerable number of the other *lymph glands* of the body usually undergo tubercular changes in the manner described. On the head, the glands usually affected are those of the larynx and the lymph glands of the parotid region; on the neck, the upper (retropharyngeal), middle, and lower cervical glands; in the anterior extremities, the glands of the shoulder [glandulæ cervicales superficiales hom.], prepectoral glands [glandulæ axillares], and brachial glands; in the posterior limbs, the inguinal glands, iliac glands [glandulæ iliacæ externæ hom.], and popliteal glands; on the udder, the pudic glands (supramammary glands); on the croup, the external pelvic glands; in the thoracic cavity, the intercostal and sternal lymph glands, and the bronchial and mediastinal glands; in the abdominal cavity, the glands of the mesentery, lumbar region, liver, spleen, kidneys; etc. The affected glands may sometimes attain a very large size.

4. In the *abdominal cavity*, besides the implication of the peritoneum (parietal membrane, omentum, and mesentery) and lymph glands, we may have tubercular changes in various other organs, such as the liver and spleen, which will then contain small and large tubercles, and caseous foci. Tuberculosis of the spleen commences usually with a uniform swelling of the Malpighian corpuscles (follicular hyperplasia). The tubercles themselves develop in the lymph follicles and in the perivascular

connective tissue. The kidneys not unfrequently suffer from tuberculosis, which causes them to become enlarged and often intermingled with numerous yellowish-white tubercles varying in size from a millet seed up to a pea. These tubercles undergo in the centre caseous disintegration and become surrounded by a capsule of connective tissue. According to Schütz, their development takes place in the interstitial tissue, during which process the normal component part of the kidneys becomes destroyed. At the same time, parenchymatous or hæmorrhagic nephritis may be present. The ovaries frequently become enlarged into tumours the size of a man's head. Ulcers are found in the mucous membrane of the affected uterus, which may, as a consequence of the disease, assume large dimensions; the nodules being situated either in the subserous intermuscular or submucous connective tissue and may break through into the uterine cavity. Sometimes we find a tubercular degeneration of the placenta. In some cases, the Fallopian tubes become closed by tubercular proliferations. Tubercular processes occur also in the vagina. In the testicles, tubercular nodules of greater or less size and caseous foci may be present. We also find tuberculosis of the tunica vaginalis combined with hydrocele. The spermatic cord, prostate, and the spermatic ducts may be implicated. The mucous membrane of the intestine, especially of the colon, sometimes shows tubercular ulcers, which are less frequently observed in the abomasum or omasum (Johne). Tubercles may become developed under the mucous membrane and serosa of the stomach and intestines.

5. Tuberculosis of the *udder*, in cases of chronic general tuberculosis, appears as a diffuse, firm enlargement, in which case, according to Bang, the gland lobules, on *post mortem* examination, appear swollen, uniformly grey coloured, and studded with small yellow points and striations, and small hæmorrhages. The larger milk ducts contain yellowish, caseous masses full of bacilli. Later on, we find in the udder a considerable increase of connective tissue (chronic, localised, interstitial mastitis) and firm tubercles, which are of various sizes and which become caseated or calcareous. Consequently the udder feels nodular, firm (sometimes almost as hard as a stone), and may attain an unusually large size, weighing up to 20 kilogrammes or more. In the wall of the milk reservoirs and larger milk canals, numerous miliary tubercles are present. Enormous deposits of tubercle bacilli may be found everywhere in the udder. All the lymph glands lying above the posterior

region of the udder become enlarged in every case, and may become indurated or caseous.

6. Tubercular changes are met with in the *brain* and *spinal cord* more frequently than is usually believed. Semmer, for instance, observed 4 cases of cerebral tuberculosis among 40 tuberculous cows. Tubercles, mostly of different ages, and varying in colour from grey to yellow, and in size from a millet seed up to that of a hen's egg, may form in the pia mater, arachnoid, brain substance, and inner wall of the ventricles. These tubercles are most commonly found in the pia mater at the base of the brain, where they may remain for a considerable time without producing any further inflammatory changes; although, not unfrequently, they give rise to inflammation of the membranes of the brain (leptomeningitis basilaris tuberculosa). The membranes of the brain may become adherent to each other and hydrocephalus internus may ensue. Sometimes the tubercles unite so as to form large masses. Similar changes manifest themselves in the spinal cord, in which the pia and dura mater become stuffed with numerous small tubercles, and frequently become adherent to each other. Even superficial ulcers, caused by pressure, have been observed on the wall of the spinal canal (Johns).

7. We sometimes meet with tubercular changes in the *eyes* (tuberculosis of the iris and choroid, and conversion of the bulb into a caseated or granulating mass); *muscular system* (miliary and large tubercles of the muscles of the skeleton, especially in the muscles of the croup, abdomen, chest, and heart, with adhesions of the heart to the pericardium); and in the *bones* (petrous portion of the temporal bone, frontal bone, occipital bone, cervical vertebræ, ribs, sternum, and the large hollow bones). Generally, tuberculosis of the bones starts from the medulla (osteomyelitis granulosa), and runs its course by the formation of reddish-grey granulation foci and lacunar breaking down of the bone tissue (caries), with central caseation and formation of cavities. Tubercular processes also occur in the cartilages (septum nasi, concha auris, etc.); in the joints (synovitis pannosa tuberculosa in the hip, stifle, knee, etc., and periarthritides tuberculosa); in the large blood vessels, as for instance, the wall of the aorta; and in the skin (tubercles, abscesses, and ulcers.)

The state of nutrition, especially in cattle suffering from tuberculosis of the serous membranes, is often good, notwithstanding the existence of extensive tubercular changes. There

is always, however, considerable emaciation and anæmia, and finally hydræmia, in cases of advanced tuberculosis of the lungs.

**Statistics.**—The researches made from 1888 to 1889 in the German Empire, respecting the seat of tubercular changes, show the following results:—One organ only was affected in from 50 to 60 per cent. of the cases; one cavity of the body, in 13 to 17; several cavities, in 15 to 20; and the flesh, in about  $\frac{1}{2}$  per cent. General tuberculosis was present in 10 per cent. The following table gives the comparative frequency of the disease in different organs:—

1. Lungs . . . . .	75 per cent.
2. Visceral pleuræ . . . . .	55 „
3. Peritoneum . . . . .	48 „
4. Costal pleuræ . . . . .	47 „
5. Bronchial and mediastinal glands . . . . .	29 „
6. Liver . . . . .	28 „
7. Spleen . . . . .	19 „
8. Uterus . . . . .	10 „
9. Lumbar glands . . . . .	5 „
10. Pharyngeal glands . . . . .	4 „
11. Trachea . . . . .	3 „
12. Udder . . . . .	1 „
13. Intestines . . . . .	1 „
14. Ovaries . . . . .	1 „
15. Lymph glands of the liver . . . . .	1 „
16. Lymph glands of the thoracic and abdominal cavities . . . . .	0.9 „
17. Heart . . . . .	0.9 „
18. Kidneys . . . . .	0.7 „
19. Bones . . . . .	0.4 „
20. Internal viscera (including the brain) . . . . .	0.3 „
21. All the organs of the thoracic and abdominal cavities . . . . .	0.2 „
22. Submaxillary glands . . . . .	0.2 „
23. Diaphragm . . . . .	0.2 „
24. Stomach . . . . .	0.16 „
25. Larynx . . . . .	0.13 „
26. Muscles . . . . .	0.1 „
27. Iliac and inguinal glands . . . . .	0.06 „
28. Brain . . . . .	0.04 „
29. Spinal cord . . . . .	0.03 „
30. Tongue . . . . .	0.01 „
31. Thymus gland . . . . .	0.01 „
32. Vagina . . . . .	0.01 „
33. Testicles . . . . .	0.01 „

**Pathogenesis.**—R. Ostertag has utilised the records of the Berlin abattoir for procuring exact information on the pathogenesis of tubercular changes in the individual organs of cattle, as compared to those of pigs, and for obtaining a definition of local and general tuberculosis. According



to these researches, the primary affections in cattle take place, most frequently, from the respiratory apparatus and from the uterus. Tuberculosis of the pleuræ and peritoneum may, however, often occur primarily without any previous tuberculosis of the lungs or intestines, on account of the tubercle bacilli passing by the lungs and intestines, without producing any tubercular changes in these organs. A primary tuberculosis of the uterus may spread through the Fallopian tubes to the peritoneum and through the diaphragm to the pleuræ. Primary peritoneal tuberculosis does not affect the parenchymata of the abdominal organs, with the exception of the uterus (Fallopian tubes) and the liver (portal region). It generally occurs in the anterior parts of the abdominal cavity, and then spreads in the direction of the lymph passages which proceed from the abdomen from rear to front. These lymph passages pass from the inferior and lateral abdominal wall, through the diaphragm, into the thoracic cavity. They first of all proceed to the posterior, then to the anterior mediastinal glands and to the bronchial glands, from which the pleuræ may also become tubercular. On the other hand, the process does not pass from the pleuræ to the lungs, with the exception of a small part of the *hilum pulmonis*. The primary tuberculosis of the pleuræ cannot spread directly to the peritoneum on account of the opposition offered by the lymph stream which flows from rear to front. The parenchyma of the lungs also remains free. Primary tuberculosis of the lungs ends either directly in tuberculosis of the pleuræ or at first attacks the lymph glands (bronchial glands) and from thence spreads to the pleuræ. Primary intestinal tuberculosis may also directly set up tuberculosis of the peritoneum or at first may affect the glands (mesenteric), and from thence proceed to the peritoneum. Besides this, primary intestinal tuberculosis may, without the instrumentality of the systemic circulation and without generalisation, lead to tuberculosis of the liver, if bacteria have penetrated through the portal vein into, but not beyond, the liver. If tuberculosis becomes general from any of the above mentioned primary foci, the comparative implication of the organs which are affected in cattle are somewhat as follows : The lungs in 100 per cent. ; pleuræ and peritoneum in 90 ; liver in 85 ; oral and pharyngeal cavities, and the intestinal canal in 60 ; the spleen in 50 ; the kidneys in 30 ; and the bones in 5 per cent. In female animals, tuberculosis of the uterus is met with in 65 per cent. ; tuberculosis of the udder in from 5 to 10 ; and tuberculosis of the ovaries in 5 per cent. The sexual organs of males are much less frequently affected than those of females.

Rieck reports with regard to the statistics of the Leipsic abattoir (67,000 cattle), that in 80 per cent. of all cases of tuberculosis, the lungs and their glands were alone implicated, and that the disease was confined to the serous membranes and their glands only in 10 per cent. General tuberculosis was found in 3 per cent. The respective percentages in the various organs were as follows : lungs, 100 ; liver, 83 ; intestinal canal, 73 ; serous membranes, 57 ; kidneys, 52 ; flesh (muscles), 49 ; spleen, 18 ; udder, 17 ; and bones, 9. Rieck comes to the conclusion that the principal gate of entrance of the infection in cattle is the lungs, and that full-grown cattle are very seldom affected from the intestines.

#### **Diagnosis of Local and General Bovine Tuberculosis.—**

Ostertag has laid down the following principles, which are carried out in the official inspection of meat in the Berlin abattoir.

1. Tuberculosis is considered to be *local* as a rule : (a) When either the peritoneum or the pleuræ, with their respective lymph glands, are alone affected. (b) When one or more organs of the same cavity of the body, together with their serous membranes, are diseased, as for instance, the lungs and pleuræ ; intestinal canal or uterus, and its respective peritoneum ; intestinal canal and liver ; and finally when the intestinal canal, liver and uterus, in addition to the peritoneum, are affected, without other organs, especially the lungs, being implicated. (c) When the affections mentioned under (b) are followed by tuberculosis of the pleuræ.

2. Tuberculosis is *general* : (a) When, besides the affection of the pleuræ or of the peritoneum, or of both membranes, diffuse miliary tubercles are present in an organ, as for instance, the lungs. (b) When, in addition to the morbid condition of these two serous membranes, limited embolic foci are found in more than one organ, as for instance, in the lungs and liver, or lungs and uterus.

**Symptoms.**—Tuberculosis of cattle is characterised by a very gradual development and by a very slow and chronic course. The initial stage of the affection escapes observation, and only in exceptional cases have signs of fever been noted as a first symptom. The symptoms vary according to the seat of the disease.

1. *Tuberculosis of the lungs* manifests itself, first of all, by a weak, dull, short cough, which, in the early stages of the irritation that gives rise to it, occurs in short jerks ; but, later on, in spasmodic and very distressing paroxysms. This cough is usually dry, and is seldom accompanied by expectoration. It is most severe in the early morning after getting up ; after exercise ; and after drinking. Respiration is more or less difficult and accelerated. With advanced destruction of the lungs, the animal pants, the ribs heave, and the nostrils remain dilated. Discharge from the nose is rare, as a rule. Sometimes, owing to dilatation of the bronchi, or breaking through of cavities of the lungs into the bronchi, a considerable quantity of a muco-purulent or caseous substance is discharged by coughing, in which cases the exhaled air has a bad smell. It often happens, if the sides of the thoracic wall or withers are pressed, that the animal will groan and cough. Percussion sounds are in many cases normal. If, however, tubercular changes spread over the surface of the lungs, and if they are sufficiently extensive, the percussion sound is dull on certain circumscribed spots, although usually it is only weak. The vesicular respiratory murmur on auscultation is generally weak, or it may be altogether absent, according to the site of dulness. In other cases, it is sharper than usual ; or we may hear râles (bronchitis), or, more rarely, bronchial respiration (cavities of the lungs and bronchiectasis).

If the disease has lasted for some time, the *state of nutrition* will usually be bad. The hair loses its gloss and becomes coarse; and the skin inelastic and hard to the touch, so that it can only with difficulty be pinched up into a fold between the finger and thumb ("hidebound"). The appetite gradually decreases, and digestive derangement ensues, on which account the secretion of milk diminishes very considerably. In some cases, we find the characteristic aspect of a chronic incurable flatulence (tuberculosis of the mediastinal glands with compression of the œsophagus). Other cases are characterised by increasing difficulty in swallowing, with salivation, nasal discharge, and shortness of breath (tuberculosis of the retro-pharyngeal glands), or dyspnœa with râles and stertorous breathing; extended position of the head and the neck, and increased sensibility and swelling of the larynx (tuberculosis of the larynx). Occasionally we find intermittent symptoms of colic, alternating with diarrhœa and constipation (tuberculosis of the intestines and tuberculosis of the abdominal glands); hæmaturia (tuberculosis of the kidneys); and a vaginal discharge (tuberculosis of the uterus). The symptoms are usually accompanied by enlargement and induration of the superficial lymph glands, especially the tracheal glands, subauricular glands (lymphomata of the parotid glands), upper, middle and lower cervical glands, glands of the shoulder, elbow, groin, hock, hip, udder, etc. The temperature of the body may be normal; although we far more frequently find an irregular, remittent, and even an intermittent fever up to  $41^{\circ}$  C., with a decided increase of temperature in the evening. The amount of blood in the body steadily decreases; the mucous membranes and the skin, the changes in which can be easily recognised in the neighbourhood of the udder and pudenda, become very anæmic; emaciation is excessive; the eyes are sunken; and the animal becomes increasingly languid and weak, and finally dies from diarrhœa or from general exhaustion. The course of the disease may last for months or even for years.

2. *Tuberculosis of the serous membranes* ("grape disease") in the majority of cases is not marked by any characteristic external signs. We may sometimes determine the presence of tuberculosis of the pleuræ by the fact that a dull percussion sound is obtained over an extensive area, owing to the great development of the tubercles. Sometimes, but only in a few exceptional cases, we may hear a friction sound, which, according to our own experience, is by no means so common as is

usually asserted. Tuberculosis of the peritoneum, when the genital organs are attacked, may manifest itself by peculiar sexual symptoms, which in cows are those of nymphomania. The animals frequently come on heat and usually remain so for an abnormally long time. They become very excited, mount their fellow cows, and do not become fecundated by taking the bull. Pregnant cows abort so frequently that abortion is in many cases the first symptom of pleural and peritoneal tuberculosis in a herd (Roloff). The general condition often remains good for a long time. We frequently find in this form of tuberculosis, the accessory symptoms mentioned in the description of tuberculosis of the lungs, especially emaciation and cachexia in the later stage of the disease. We may sometimes demonstrate the presence of peritoneal "grapes" on the rumen and on the portion of the peritoneum which lines the wall of the abdomen, by palpation of the hollow of the flank (Kleinpaul).

3. *Tuberculosis of the brain* may occur as an independent primary affection, and in most cases, is a complication of tuberculosis of the lungs. Its course is generally characterised by symptoms of an acute leptomeningitis. The animal shows great excitement and suffers from severe attacks of fury, convulsions and spasms, which often closely resemble those of cerebro-spinal meningitis, or which may have an epileptiform character. Later on, these symptoms alternate with unconsciousness, stupor and paralysis. It often happens that the patient collapses suddenly. In other cases we may observe symptoms of affections of the nerve centres, such as staggers (rotatory and circular movements); oblique carriage of the head (tuberculosis of the middle ear); paralysis of the facialis, oculo-motorius, opticus, and trochlearis; hemiplegia; etc. Periodical attacks of excitement may sometimes be noticed. Tuberculosis of the spinal cord manifests itself by a heavy tread, high action, and finally by paralysis of the loins.

4. *Tuberculosis of the udder* may appear as a first and single symptom of tuberculosis in animals which are otherwise quite healthy, although it is more often secondary. According to Bang, it manifests itself as a diffuse, painless, and comparatively firm swelling, usually of one quarter (one of the posterior quarters as a rule) of the udder; and more rarely, of two quarters. The milk, contrary to what is the case in other inflammatory conditions of the udder, is at first normal; but it becomes in about a month thin and watery,<sup>§</sup> mixed with flakes, and sometimes,

though not always, it contains bacilli. We ourselves have frequently searched in vain for bacilli in the milk of cows which were thoroughly infected with tuberculosis. Bollinger proved that milk from the udder of tuberculous cows was undoubtedly infectious in 55 per cent. of the cases; although the demonstration of tubercle bacilli in such infected milk succeeded only once in 20 cases. The swollen parts of the udder become more and more indurated, until at last they are as hard as a stone. The process frequently spreads from the posterior quarters to the anterior quarters of the udder. The supramammary lymph glands (pudic glands) often become enormously enlarged.

5. In *general tuberculosis*, the morbid changes described respectively under the previous four sections are combined in various ways. Besides, we may also find swellings of the bones and joints (*tumor albus*), stiffness and lameness (tuberculosis of the bones), as well as the before-mentioned tubercular changes of the skin and eyes. In very rare cases the disease suddenly assumes a peracute and fatal course (acute miliary tuberculosis).

**Clinical Diagnosis**, as compared to diagnosis by inoculation or by bacteriological methods, is very uncertain; because tuberculosis does not possess characteristic symptoms. No diagnostic sign may be present, especially during the first few months of the disease. Even in the more advanced stages of the disease, an exact diagnosis from purely clinical data can seldom be made. This fact, which is denied without sufficient reason by some, is of great practical importance in forensic veterinary medicine; because, from a legal point of view, the proof of the existence of tuberculosis necessitates as a rule, the slaughter of the animal. On the whole, the following clinical factors are of importance in the event of a diagnosis, *intra vitam*, being required.

1. *For tuberculosis of the lungs*: The simultaneous occurrence of severe disturbances in nutrition, with symptoms of lung affection (coughing and the respective results of percussion and auscultation), and the enlargement of the external lymph glands, especially in cows and heifers.

2. *For tuberculosis of the serous membranes*: Increased sexual desire, sterility, tendency to abortion, friction sound (on rare occasions), and more or less extensive dulness. Here, perhaps, a knowledge of the breed may help us to arrive at a

correct decision. The presence of tubercular proliferations of the peritoneum may be proved by introducing the hand into the rectum or by pushing the hand under the last false ribs. We may also mention incurable chronic flatulency with normal digestion.

3. *For tuberculosis of the brain* : Symptoms of acute inflammation of the brain, complicated with lung symptoms, glandular swelling, and loss of condition.

4. *For tuberculosis of the udder* : The highly characteristic, at first diffuse, firm and painless swelling ; subsequent changes in the milk ; induration of the udder ; enlargement of the lymph glands ; and proof of the presence of the bacillus in the milk by inoculation.

5. Besides being aided by proof of chronic tympanites taken in connection with symptoms of pulmonary tuberculosis, our diagnosis of *tuberculosis of the mediastinal glands* will, according to Albrecht, be facilitated by the introduction of an œsophageal tube. He states that, immediately after the removal of the gas by means of this tube, normal digestion reappears for a shorter or longer period in cattle, contrary to what takes place in non-tuberculous cattle.

Up to the present, no practical experiments have been made with the ophthalmoscope for diagnosing tuberculosis of the choroid and iris. Röbert recommends, for facilitating the diagnosis, covering over, with a cloth, the nostrils and mouth of cattle which are being examined, in order to oblige them to adopt forced breathing, so that any abnormal respiratory sounds may become more easily heard. In a similar manner, an injection of 0.3 gramme of pilocarpin, by inducing forced breathing, will enable us to hear a well-marked friction sound in cattle suffering from tuberculosis of the serous membranes (Walther). Peuch states that we can make an exact diagnosis by inserting a seton and inoculating guinea-pigs with the produced pus.

**Bacteriological Diagnosis.**—The presence of the bacilli is constant in the tubercular sputum of men ; but only in a small percentage of cases in that of cattle. Hence, in the latter, the bacteriological examination of the sputum is by no means so important, from a practical point of view, as in the former. For the same reason, the method proposed by Pöls, of obtaining mucus for bacteriological examination, by inserting a cannula between two of the tracheal rings, is frequently

without result. For instance, in the case of a cow which was suffering from fully-developed tuberculosis, we succeeded by this procedure in obtaining bacilli only after repeated experiments; and in only one single streak preparation were able to demonstrate the presence of a few tubercle bacilli. Nocard's plan of obtaining mucus from the trachea, by injection of veratrine or eserine, is just as uncertain. It is frequently so difficult to find the bacillus, even in sections of the tubercles, that a large number of preparations have almost always to be examined.

Milk gives better results for bacteriological examination. It sometimes contains a considerable number of tubercle bacilli, the presence of which can be microscopically demonstrated by staining (see further on). It seems, however, that the milk of tuberculous cows contains spores more frequently than bacilli. As these spores cannot be stained, they escape microscopical examination; for which reason the bacteriological demonstration of milk is uncertain. We have failed to obtain bacilli in milk, even when the udder was gravely affected. Bollinger found that among 20 tuberculous cows the milk was infectious in 11 cases; but that the presence of the bacilli could be proved in only one of them. Gaffky states that the bacteriological examination of *fæces* affords valuable diagnostic results.

In those cases which cannot be decided by the microscope, the only certain means is the inoculation of milk and other suspected material, such as vaginal secretions, uterine discharges, excised lymph glands, etc. According to the experiments of Arloing, Bollinger, Verneuil, and others, guinea-pigs give better results for this purpose than rabbits. For inoculation, it is best to introduce a minute quantity of the infectious material into the abdominal cavity, in which case a general miliary tuberculosis with characteristic anatomical and microscopical conditions will become developed in 10 or 12 days. Hirschberger recently succeeded in transmitting tuberculosis to guinea-pigs in 33 per cent. of the inoculations he made with the milk of cows which were suffering from tuberculosis only in the lungs. A nearly similar percentage (37.5) was obtained by Ernst in cases which presented no symptoms of disease in the udder. Bang obtained a smaller percentage. Experimental inoculation of guinea-pigs and microscopical examination must consequently be considered to be the best means of recognising tuberculosis in the living animal.

**Microscopic Demonstration of the Tubercle Bacillus.—**

1. According to Ehrlich and Koch, the material to be examined (mucus, milk, pus, etc.) should be spread out into the finest possible film upon a cover glass. In the case of milk, Johne advises that it should be diluted with from 2 to 5 times as much water, and mixed with sufficient acetic acid to precipitate the albumen and bacilli. The precipitate is then placed on the cover glass, and is allowed to dry in the air, after which the cover glass is passed several times slowly through a gas flame. As soon as it is cool it is floated, with the film side downwards, upon the cold staining solution, which is described in detail further on, and in which it is allowed to remain from 12 to 24 hours. According to our experience, the cover glass should remain in the cold staining solution this length of time, and not, as formerly stated, from half to one hour. A better plan, as we have found, is to place the cover glass not merely in the staining solution, but to heat both together over a gas jet until perceptible steam is given off; and after they have been allowed to cool, to repeat the process once or twice in a similar manner. By this more expeditious method we obtain better preparations. The staining solution is made in the following way: 5 c.c. of aniline oil are mixed with 100 c.c. of distilled water and filtered. To 100 c.c. of this aniline water are added 11 c.c. of an alcoholic solution of methyl violet, or fuchsin, and 10 c.c. of absolute alcohol. This solution should be renewed every 10 or 12 days. After the cover glass has been taken out of the staining solution, it should be rinsed in water and placed for a few seconds in dilute nitric acid (1 to 3), by which it is decolorised, and it must then be again thoroughly rinsed in water. By means of this method of decolorisation, the tubercle bacillus alone retains the stain. The cover glass is finally put for about five minutes in a 1 to 2 per cent. watery solution of Bismarck brown or malachite green, rinsed, dried, placed in Canada balsam, and put under the microscope. By this method the tubercle bacilli are stained dark-blue or red, whilst all other bacteria and cells appear brown or green.

2. Weigert takes a 2 per cent. solution of gentian violet, and adds a  $\frac{3}{4}$  per cent. of liquor ammoniæ and 10 per cent. of absolute alcohol. If the preparations be exposed in this solution for 30 minutes to a temperature of 40° C., they can be decolorised in a 33 per cent. solution of nitric acid for a long time, and even until they completely lose their colour, without impairing the deep blue-black staining of the bacilli.

3. Gabbet has published a new staining method, which, he states, is the most convenient one in practice. He places, for two minutes, the dry preparation, which is made in the usual way, in a fluid composed of 100 grammes of a 5 per cent. aqueous solution of carbolic acid and 10 grammes of absolute alcohol, in which 1 gramme of fuchsin has been dissolved. Directly afterwards he puts it for one minute in a mixture of 100 grammes of a 25 per cent. solution of sulphuric acid, in which 2 grammes of methylene blue have been dissolved. It is next rinsed in water and examined in this medium, or still better, it is rinsed in absolute alcohol and mounted in Canada balsam. The bacilli will then appear red on a blue ground. If a more intense staining of the bacilli be required, the first solution is heated for two minutes until steam arises from it.



4. According to Ziehl-Nelsen, the preparation to be examined is placed in the usual way upon a cover glass, is passed through the flame, and is then immersed in a solution of carbolic acid and fuchsin, which is heated over a Bunsen's burner in a hollow ground slide on a ring of wire netting. The said solution consists of 1 part of fuchsin, 5 parts of carbolic acid, 10 of absolute alcohol, and 95 of distilled water. As soon as the solution begins to give off steam, the preparation is taken out and placed first of all in a 5 per cent. solution of sulphuric acid, then in a 70 per cent. solution of alcohol, and finally in a solution of 2 parts of methylene blue in 100 parts of water. The preparation is then washed in water and placed upon the slide.

5. Czaplewski moistens with the carbolic fuchsin solution the previously prepared cover glass preparation, which has been got ready in the usual way, and holds it over the flame till it gives off vapour. On draining away the carbolic fuchsin solution, the cover glass is dipped from six to ten times into a concentrated alcoholic solution of yellow fluoresceine. It is then dipped ten or twelve times in a concentrated alcoholic methylene blue solution, and finally rinsed in pure water.

6. The bacilli are coloured as follows by the Löffler-Fränkel method, which Kitt considers very simple and convenient. Löffler's aniline water fuchsin solution is made by preparing, in the usual way, 100 c.c. of aniline water by shaking aniline oil with distilled water and filtering through previously moistened filter paper. To this is added 1 c.c. of a 1 per cent. solution of caustic soda, which renders alkaline the neutral aniline water. It is then poured into an Erlenmeyer's flask, or, just as well, into a medicine bottle, to which is added 4 to 5 grammes of solid fuchsin. The bottle is closed with an india-rubber plug or cork, and should be frequently shaken before use. One drop of this solution is placed upon the film side of a cover glass, which is then held in a forceps over a spirit or gas flame at such a height that the fluid upon the glass becomes gradually heated and steam rises in two to five minutes. The cover glass is then dipped into the solution, which causes the differentiating or double staining. This solution, according to B. Fränkel, is a mixture of 50 c.c. of alcohol, 30 c.c. of water, 20 c.c. of nitric acid, and as much methylene blue as will dissolve in it after repeated shaking. The solution, which we can easily prepare ourselves, is filtered into a medicine bottle, and can be kept in stock. A few cubic centimetres are put into a flat glass dish, the drop of Löffler's fuchsin which had been placed on the cover glass is poured off, and it is then placed immediately into the blue acidulated mixture for 1 or 2 minutes. It is then rinsed in water and placed upon the slide. The glass is dried and mounted in Canada balsam in the usual way. The tubercle bacilli are thus stained a pure red, the rest being blue. It is not necessary to prepare a fresh solution for each tubercle bacilli staining; for we can keep in stock, in two separate medicine bottles, the two solutions, namely, one for staining the bacilli, and the other for double staining. We further require only two glass capsules and can undertake the staining and heating of the cover glass preparation by holding it over a flame. If preferred, we can place the cover glass in the fuchsin capsule under the filter and then hold it over the flame until the appearance of steam. The time required for staining is only a few minutes. Good

ingredients, practice, and skill are necessary with all these methods of staining.

**Diagnostic Inoculations in Guinea-pigs.**—Successful results are obtained quickest by intraperitoneal inoculations. Numerous tubercles of the size of a millet seed form 10 or 12 days after the injection. Subsequently we may find many caseous tubercles varying in size from a pea to a bean, in the omentum and in the enlarged spleen; and small yellow nodules in the liver. If a subcutaneous inoculation be made, a nodular induration will develop at the site of the injection. In the second week, a suppurating ulcer forms, and from the second to the third week the neighbouring lymph glands swell to the size of a hazel nut. After the sixth week, marked emaciation takes place. If a *post mortem* examination be made from 6 to 12 weeks after the inoculation, we shall find that the tubercles at the site of the injection and the neighbouring lymph glands are caseous. Numerous tubercles and caseous foci are found in the mesentery, omentum, liver, and in the greatly enlarged spleen. Occasionally miliary tubercles become developed in the lungs.

**Tuberculin as a Diagnostic Agent.**—Tuberculin, which was discovered by R. Koch in 1890, and which is a glycerine extract of pure cultivations of tubercle bacilli, has, in human medicine, proved to be neither a remedy nor a diagnostic agent. On the other hand, the results of numerous inoculations of tuberculin in cattle demonstrate the fact that, tuberculin is a very valuable, although not absolutely infallible aid to the diagnosis of bovine tuberculosis. From experiments made in many places, we may deduce the following rules for tuberculin inoculations:—The dose of tuberculin for horned cattle is 0.5 gramme. Smaller doses are sometimes sufficient, although the reaction they produce is often too weak and too fugitive. The reaction in tuberculous animals consists chiefly in a rise of temperature (inoculation fever or reaction fever) which appears in from 12 to 15 hours after the injection of the tuberculin. If an increase of temperature of at least  $1.5^{\circ}$  C. is found in an animal after the inoculation, the presence of tuberculosis may be assumed as probable. If, before inoculation, the temperature of the bovine patient be abnormally high, especially if it be  $39.5^{\circ}$  C. or more in the rectum, no reliable deduction can be made. We must also point out that the reaction, as a rule, is inversely proportionate to the extent and duration of the disease, so that excessively tuberculous cattle show less increase of temperature than those with slight localised tuberculosis. Hence, in severe and old standing cases of tuberculosis, tuberculin is not very reliable. One

inoculation generally suffices. In doubtful cases it may be repeated after a few weeks. The evening is the best time to make the inoculation; for then the rise of temperature can be observed during the following day. The temperature must be taken at least four times after the inoculation, namely, 9, 12, 15, and 18 hours after injection, prior to which the temperature should be taken at least twice, that is to say, six hours before, and directly prior to the injection of the tuberculin. The side of the neck is the best site for the inoculation. Tuberculin has, only in a very few cases, caused injury to the inoculated animals (acute miliary tuberculosis, fatal intestinal hæmorrhage, œdema of the lungs, temporary sterility, and contraction of the uterus). The quantity of the milk becomes diminished during the time of the reaction, to the extent of a few quarts, in consequence of the resulting fever and loss of appetite. The milk secreted during that short period should not be used for human food.

In several cases, beasts which had reacted to tuberculin have, on being subsequently slaughtered, proved free from tuberculosis. Such failures in diagnosis have been estimated at 15 per cent. in Germany and at 4 per cent. in Denmark. Cattle suffering from the following diseases sometimes react in the same way as those that are affected with tuberculosis: actinomycosis, botryomycosis, abscesses of the lungs and liver, abscesses following traumatic pericarditis, inflammation of the udder, caseous echinococci, distomatosis, diseases due to pulmonary worms, emphysema of the lungs, and chronic diarrhœa. We may therefore conclude that tuberculin is not exclusively a reagent for tubercle bacilli; and that it performs a similar office for the streptococci of pus and other micro-organisms. As it is not a specific diagnostic agent for tuberculosis, its reaction cannot be accepted in forensic veterinary cases as a positive proof of the presence of tuberculosis; although the fact of the reaction occurring would strongly suggest such a probability. Tuberculin, however, may be used with advantage for diagnostic purposes, when we wish to select animals for breeding, and as a prophylactic for combating tuberculosis in large herds.

**Differential Diagnosis in Cattle.**—1. Tuberculosis of the lungs may be clinically mistaken for disease of the lungs due to echinococci, after-conditions of pleuro-pneumonia, and verminous bronchitis. Tuberculosis of the serous membranes

may be mistaken for pleuritis ; tuberculosis of the mediastinal glands, for ordinary chronic tympanitis ; and tuberculosis of the uterus, for chronic metritis. Tuberculosis of the brain is frequently mistaken for simple inflammation of the brain, cerebro-spinal meningitis, rabies, malignant encephalitis (Zündel), staggers, etc. Tuberculosis of the lymph glands may also be easily mistaken for actinomycosis, leucæmia, and general sarcoma. There is no doubt that many reported cases of tuberculous lymphomata of the pharyngeal cavity, region of the parotid glands and oral cavity, and of tuberculosis of the tongue and udder, were nothing else than actinomycosis. A complication of tuberculosis and actinomycosis sometimes occurs. In the majority of such cases, no positive conclusion can be arrived at without making a section for microscopical examination.

2. The following diseases may be mistaken for tuberculosis ; pleuro-pneumonia contagiosa (the lungs) ; actinomycosis (mouth, pharynx, lymph glands, udder, and bones) ; leucæmia (liver and kidneys) ; amyloid degeneration (liver, spleen, and kidneys) ; metastatic abscesses and echinococci (lungs, liver, spleen, and kidneys) ; bronchial pneumonia ; pneumonia due to foreign bodies ; bronchitis ; peribronchitis ; gastro intestinal ulcers ; etc.

Special attention has been lately directed to the discrimination between tuberculosis of the liver and echinococcus multilocularis. The disease, set up by echinococcus m., is distinguished, according to Ostertag, from tuberculosis by the fact that it does not cause a simultaneous affection of the corresponding lymph glands ; by the fact of the affected part being elastic ; and, as a rule, by the eruption of fresh vesicles on its border.

**Prognosis.**—Tuberculosis in cattle is curable only during the very early stages and when it is local. In all cases, the prognosis is very unfavourable, and consequently the best thing to do is to slaughter the animal as soon as possible.

**Therapeutics.**—As it is futile to treat tuberculosis by medicine, our efforts should be directed to the application of prophylactic measures, the chief of which are : supporting the strength by a rational system of food and management ; separation of the healthy ; early slaughter of the sick ; exclusion of affected animals from breeding ; prohibition of the use

of raw milk ; and disinfection of the sheds. It is still an open question whether veterinary police regulations ought to be enforced against tuberculosis. As regards this subject, Lydtin proposed the introduction of compulsory notification, publication of cases, slaughter of affected and suspected animals, and police supervision of infected sheds ; that indemnification for slaughtered animals should be given ; and that penal regulations should be proclaimed. We venture to doubt the necessity as well as the possibility of practically carrying out these sweeping measures. The notification of cases is impracticable on account of the difficulty and the impossibility of making a positive diagnosis *intra vitam*. The slaughter of all cattle affected by tuberculosis would demand very extraordinary sacrifices, owing to the great dissemination of the disease. We need consider only the following practical rules :—(1) Tuberculous animals should be slaughtered as early as possible, with reciprocal insurance or indemnification. (2) Early slaughter of the calves of tuberculous cattle. (3) Destruction of the tubercular parts. (4) Boiling of milk.

We may mention as a forensic fact that tuberculosis is, in some countries, considered to be an unsoundness which may be covered by special warranty. Thus, we find the period of warranty to be 28 days in Bavaria, Württemberg, and Hesse for "grape disease," and 14 days for bovine pleuro-pneumonia ; and 8 days in Prussia, 50 in Saxony, 30 in Austria, and 20 in Switzerland for tuberculosis.

**Milk Hygiene and Official Inspection of Meat.**—The identity of tuberculosis of cattle with that of man has been proved by numerous positive transmission experiments ; by the uniformity of the anatomical structure of the tubercular changes ; and, above all, by the positive proof that the same bacilli exist in both forms of the disease. Gerlach was the first to point out in Germany that cases of transmission of tuberculosis from cattle to man were by no means rare. The tuberculosis of cattle, and in fact that of all other animals, is consequently of the greatest importance to human therapeutics from a sanitary police point of view. Here we have, in particular, to consider the question of the suitability of the milk and the flesh of tuberculous animals for human food.

1. The unboiled milk of tuberculous cows must be regarded as injurious to health. According to the experiments of Bang, tuberculous milk becomes a safe food only after having been heated to 85° C., which temperature destroys the bacteria. It is almost needless to say that the milk of tuberculous or suspected animals should be used only after boiling, which should be the rule in all cases ; for the milk of even apparently healthy cows may contain tubercle bacilli.\* The drinking

\* See also page 179.

of milk warm from the cow, which was formerly recommended as a remedy against human tuberculosis, is to be condemned under all circumstances. According to Friis, the dilution of the milk of commerce with water has no perceptible influence in decreasing its contagiousness. Infection may also be transmitted by the consumption of products (cheese, whey, butter, etc.) made from tubercular milk (Galtier, Heim, Laser, Bang, Roth, and others).

2. The flesh of tuberculous cattle, as already stated, is less infectious than the milk, and can consequently be used as food under certain precautions. Intraperitoneal inoculations of guinea-pigs with muscle juice obtained from 12 beasts which had suffered in various degrees from tuberculosis of the serous membranes, showed a negative result in all the 16 guinea-pigs which had been inoculated (Bollinger). In spite of this, it is advisable to eat the flesh of tuberculous animals only after it has been well boiled. Salting, pickling, and smoking the flesh offer, according to the researches of Forster, no protection whatever against infection. The German sanitary police regulations of the present time state generally that the meat must not be used as food in the following cases: 1. In general tuberculosis, that is, in "the presence of acute miliary tubercles, or of chronic tubercular infiltrations in two or more organs which are situated in different cavities of the body, and which are not connected with one another either directly or through the lymphatics, pulmonary blood vessels, or portal vein, but only by the general circulation of the blood" (*Order of the Prussian Minister*, 15th September, 1887). 2. When it is stuffed with tubercles in the serous membranes. 3. When great emaciation takes place, while only local foci are present, or when, during the life of the animal, the symptoms of a feverish general disease have manifested themselves. 4. When the meat is in a nauseous condition. On the other hand, the meat can be used as food in cases of localised tuberculosis under the proviso that the diseased organs have been destroyed. Ostertag (*Handbook of Inspection of Meat*, 1895) lays down the following rules:—

a. All tubercular organs and even all organs in which only the lymph glands are affected with tuberculosis must be excluded from consumption as being dangerous to health; for this reason all the lymph glands at the respective entrances of the digestive canal and respiratory tract must be examined in every slaughtered animal.

b. Even when only a few foci are present in an organ, the whole organ is to be regarded as dangerous to health. A tubercular organ cannot, like one which contains animal parasites, be rendered fit for food by the removal of the affected parts.

c. The flesh of animals with slight, local tubercular changes may be passed as fit for sale after the removal of the tubercular foci.

d. The flesh of animals which are affected with extensive, but doubtless, local tubercular processes, may be sold under declaration.\* The same regulation stands good in cases of a healed generalisation which is confined to the internal organs (lungs, liver, spleen, or kidneys).

e. All animals which show marked emaciation and those which

\* This declaration has reference to the institution known in Munich as *Freibank*, which is a retail meat-market where inferior meat which has been sterilised, is sold under declaration of its condition.—TR.

manifest tubercular changes in the flesh, or signs of a very recent infection of the blood (swelling of the spleen, swelling of all the lymph glands and miliary tubercles in the lungs, liver, spleen, or kidneys), should be entirely excluded from human food, and should be used only for technical purposes.

*f.* The flesh of those animals in which the local character of tuberculosis and the innocuousness of the flesh are doubtful (especially when tubercular cavities are present, and when the bodily condition of the animal begins to become affected) may be allowed to be sold under the provision that it has been thoroughly boiled, or better still, sterilised by steam.

### TUBERCULOSIS OF PIGS.

**General Remarks.**—Tuberculosis of swine, especially in young pigs, is a fairly common disease, although it is far less frequent than tuberculosis of cattle. In the abattoirs of Germany the number of tuberculous pigs was from 2 to 4 per cent. In 1891 the percentage in Prussia and Saxony was 1; and in 1894 in Saxony, 2. Among the  $3\frac{1}{2}$  millions of pigs which were killed during the years 1883 to 1892 in Berlin, 56,000 (equal to 1.6 per cent.) were found to be tuberculous. During 1892 the percentage was 2.7 in Berlin, 1 in Dresden, 2 in Leipsic, 2.4 in Lübeck, and 4 in Elbing. In Denmark from 10 to 14 per cent. of all the slaughtered pigs are tuberculous. Tuberculosis is said to be very common among pigs in Belgium.

Among pigs, which thus differ from cattle, tuberculosis is found to be most common in those that were less than 1 year old. The predisposition to the disease is greatest in improved English breeds. The chief causes which act unfavourably on the constitution of swine are: unnatural bringing up, fattening, exclusive stall-feeding, and early forcing. The commoner the breed, the less liable it is to tuberculosis. Hertwig points out that the pigs of Servia, Galicia, and the Bakony forest (Hungary), which have been but little improved in breed, seldom become tuberculous; probably because most of them are pasture-fed. The ingestion of the tubercle bacilli frequently takes place through the milk of tuberculous sows. In such a case, all the members of a litter may become affected. The respiratory air must also be considered to be a vehicle for the infection; because experience proves that all the animals of a sty often become simultaneously tuberculous. As in cattle, we must assume the heredity of tuberculosis in swine, because all the pigs of certain breeds become affected by the disease. Meyer has remarked that in castrated pigs infection may originate in the operation wound. Cattle often transmit the disease to pigs

by means of milk and waste products obtained in the preparation of cheese and butter, and especially the crude sediment left from the centrifugal process used in dairy work, and also by consumption of the tubercular parts of the dead body. It has not yet been proved, although it is probable, that infection may be transmitted to pigs by the tubercular sputum of man.

**Pathogenesis and Anatomy.**—Tuberculosis of pigs differs from that of cattle principally by the fact of its being a well-developed food-tuberculosis; as its starting point is generally in the digestive apparatus, and bacilli can only with difficulty be found in the affected organs. According to Ostertag, tuberculous inflammation is met with chiefly in the jejunum, ileum, and cæcum, with ulceration of their mucosa, and formation of tubercles in the muscularis, subserosa and mesenteric glands. At the same time, we may find tubercular tonsilitis (less frequently pharyngitis) and a diseased condition of the submaxillary, retropharyngeal, and upper cervical lymph glands. The encroachment of tubercular intestinal ulcers on a branch of the portal vein causes embolic tuberculosis of the liver, in the event of a numerous invasion of bacilli, extensive miliary tuberculosis of the liver, and consequent general tuberculosis. The tuberculosis of the middle and inner ear is of special importance. According to Schütz, it begins with catarrh of the pharynx, in which case the tubercular inflammation at first spreads through the Eustachian tube to the tympanic cavity and sets up a tubercular osteomyelitis, periostitis, and parotitis with rarefaction and necrosis of the bones. From thence the process may extend to the cerebral membranes, the cerebellum, medulla oblongata, and even into the external auditory passage, which becomes filled up by a tubercular tumour.

When, as happens only in rare cases, swine tuberculosis begins primarily in the respiratory apparatus, it takes the form of a tubercular broncho-pneumonia, chiefly at the base and apex of the lungs with consequent implication of the bronchial glands, and, less frequently, of the pleuræ. The lungs, especially on their surface, become studded with numerous miliary and sub-miliary, grey, translucent tubercles, or with larger hard tubercles which vary in size from a pea to a hazel-nut, and which undergo caseous disintegration in their centre, and gradually become indurated, being in some cases as hard as a piece of cartilage. The surface of the lungs thus presents a spotted and uneven appearance. Besides this, we may find comparatively large



caseous pneumonic foci, the formation of interstitial tissue, and chronic bronchitis, with their results. In consequence of the diseased condition of the pleuræ, the mediastinal glands and the subpleural lymph glands of the thoracic vertebræ and sternum may also become infected. We very rarely see tuberculosis of the trachea and larynx along with tuberculosis of the upper, middle, and lower cervical glands. Primary tuberculosis of the uterus is also rare. On the other hand, general tuberculosis is common in pigs.

**Statistics.**—According to Ostertag, the lungs in general porcine tuberculosis become affected in 100 per cent. of the cases; liver, in 90; spleen, in 80 to 85; oral cavity, laryngeal cavity and intestinal canal, in 80; kidneys and serous membranes, in 30; bones, in 15 to 20; and mammae, testicles, uterus, and articulations, in 1 per cent. This table shows that in pigs the spleen is much more frequently, and the serous membranes of the abdomen and thorax much more rarely affected by tuberculosis than in cattle. Ostertag lays down the following views on the diagnosis of local and general tuberculosis:—

1. Speaking generally, tuberculosis in pigs is *local*, (a) when any organ with its lymph glands, as for instance, the intestine with the mesenteric glands, is affected; (b) when in certain cases two organs, such as the tonsils and the intestine, with respectively the submaxillary and mesenteric glands, are involved, or when in intestinal tuberculosis, the liver only shows limited embolic foci; (c) when the morbid condition of the lungs, pharyngeal cavity, and intestine has not had an embolic origin.

2. Tuberculosis in pigs is *general*, (a) when disseminated embolic foci (acute miliary tuberculosis) are found along with primary affection even in only one organ, as in miliary tuberculosis of the lungs, intestines, and their respective lymph glands; (b) when, besides, the primary affection, restricted embolic foci (chronic general tuberculosis) are found in at least two organs, as for instance, in the lungs and liver along with tuberculosis of the intestines, and in the lungs or liver and uterus, etc.

**Symptoms.**—The symptoms of tuberculosis in pigs vary according to the part affected. Frequently there is no external sign of the disease.

Young pigs when suffering from *intestinal tuberculosis* are backward in growth and become gradually emaciated. The mucous membranes become very pale, and the skin covered with dark grey crusts (*Russ der Ferkel, suie des porcelets*, soot of young pigs). These symptoms are followed by various disturbances of digestion, such as vomiting, tympany, slight colic, and diarrhœa. The internal temperature is alternately high and normal. Later on, the patient steadily loses condition. The flanks are tucked up, the eyes sunken, and the belly pendulous. The adherent intestinal loops can frequently be felt from the

outside as a hard knotty mass of the size of a man's fist, or even larger. The animal sometimes manifests pain during palpation of the posterior part of the body. Death occurs from progressive general debility, usually in a few months, or even in a few weeks, if the lungs and intestines be simultaneously affected.

*Tuberculosis of the brain* in pigs runs a very irregular course. First of all we observe automatic movements, such as rearing and turning round and round; convulsions and spasms of the muscles of the trunk and limbs; nystagmus; etc. At a later stage the following paralytic affections appear: hemiplegia, unilateral paralysis of the tongue, oblique position of the head, paralysis of the muscles of the eyes, and especially of the eyelids; unilateral paralysis of the face with the snout drawn to one side; and pendant condition of the ears.

In *tuberculosis of the lungs* in pigs, the sufferer has at first a short and dry cough, which becomes, later on, very harassing, and generally comes on in choking paroxysms. The breathing is accelerated and forced, and the animal becomes gradually emaciated. The disease has a shorter course in young pigs than in old ones, and may last for weeks or even months.

The chief *diagnostic points* are: Occasional visible tubercular proliferations in the outer auditory passage; swelling of the cervical lymph glands; and eruption of tubercles in the interior of the eye (Azary). Bacteriological examination is of less importance for diagnosis in pigs than in cattle; because even in anatomical preparations the bacilli can be found only with the greatest difficulty in swine.

**Differential Diagnosis.**—As tuberculosis in pigs can, as a rule, be proved only after slaughter, we should above all things pay attention to the anatomical changes, which we should be careful not to mistake for the chronic processes of contagious pneumonia of the pig. Ostertag differentiates tuberculosis and contagious pneumonia as follows:—

1. In *tuberculosis*, changes may be found in all the organs of the body. Solid tubercles along with caseous foci may be met with in the parenchyma. The lymph glands of the tubercular organs are invariably enlarged, and caseation in them always emanates from several points, which correspond to the conglomerates of the tubercles, so that the enlarged lymph glands appear to be infiltrated by numerous small caseous foci. As a rule, calcification quickly follows caseation.

2. In *chronic pneumonia*, changes are generally found in the

lungs, and, less frequently, in the skeleton. We find in the lungs only cavities or formations of sequestrum, varying in size from a pea to a man's fist. Usually an adhesive pleuritis is also present. The lymph glands most frequently affected are the bronchial, tracheal, and inguinal glands. When attacked, they become entirely caseous and present the appearance of a round sack filled with dried caseous pus, arranged in concentric layers without any calcification. The bacteria of contagious pneumonia can be easily found in the pus foci.

It is worthy of remark that tuberculosis and chronic pneumonia may occur simultaneously in a pig, in which case the presence of tuberculosis may be proved by experimental inoculations in guinea-pigs. As already stated, the tubercle bacilli, as a rule, cannot be found. No experiments with tuberculin have as yet been made in pigs.

#### **Scrofulous or Caseous Inflammation of the Intestines.—**

The etiology of the disease of pigs which was described by Roloff under this name, has not yet been properly elucidated. It probably belongs partly to tuberculosis, and partly to swine fever, and is characterised by peculiar intestinal changes that chiefly implicate the colon, the loops of which form, by adhesion, a firm bundle. The individual intestinal convolutions represent thick eminences with divisions and cavities by which the intestine assumes a rosary-like aspect. The wall of the intestine is thickened and covered with nodular prominences, the centre of which contains caseous foci. Miliary nodules are imbedded under the serosa and the submucosa. The mucous membrane of the intestine shows circumscribed, copper-coloured hæmorrhagic foci or necrotic disintegration, with scabbing and exfoliation of the upper layers, and sometimes has broad caseous masses on its surface. Peyer's patches are swollen and ulcerated. Large tubercular ulcers, with raised and thickened margins, are found in the mucous membrane. The ileo-cæcal valve frequently projects into the cæcum in the form of a long firm plug, with a scabby discoloured surface and ulcerated margin. The intestines become sometimes narrowed by cicatrisation. The mesenteric glands become enlarged and contain caseous foci.

#### **EQUINE TUBERCULOSIS.**

**Occurrence.**—Tuberculosis is not a common disease in horses. Many of the cases which were published long ago are not altogether free from suspicion. On the other hand, it is not improbable that a few cases reported as glanders were really those of tuberculosis. In recent times, the presence of tuberculosis has been demonstrated more frequently in the horse than formerly. Bang has, for instance, collected 29 cases. In

Saxony 0.8 per cent. of 3,500 slaughtered horses were found to be tuberculous.

**Pathogenesis.**—The chief port of entrance in horses for the tubercle bacilli is the lungs; a fact which may be inferred from the frequency with which these organs participate in the disease. Also, the affection in them is often primary (Nocard and Johne). Nothing certain is known as to the source of the infection, whether it be horses, cattle or men. In some cases, transmission from cattle through cohabitation appears to take place. According to Csokor and Bang, the intestines are one of the chief points of invasion for the bacteria. Their entrance along with the food is proved by the fact that the oldest and most important tubercular changes are found in the mesenteric glands. According to Bang, the bacilli, among other things, are transferred to horses by feeding with the milk\* of tuberculous cows and by the use of straw from infected cow-sheds. It is very difficult to transmit tuberculosis to the horse by inoculation.

**Anatomical Changes.**—The changes are pretty similar to those of tuberculosis of cattle and may become localised in the lungs as well as in the serous membranes; or may become generalised by spreading simultaneously over several organs. We may find in the lungs small miliary tubercles, or large firm nodules which are caseous in their centre, and which may attain the size of a walnut. These nodules may unite into a firm mass infiltrated with yellowish foci, or may form cavities. As in cattle, the lungs may show well-marked symptoms of miliary tuberculosis (multiple embolic foci). Numerous tubercle bacilli are contained in the disintegrated caseous deposits. The bron-

\* McFadyean (*Journal of Comparative Pathology and Therapeutics*, September, 1896) draws attention to the fact "that in a considerable proportion of cases there was a distinct history of the animal having been largely fed with tuberculous milk. Now when one reflects that certainly not one horse in several hundreds is at any period of its life fed on cows' milk, the frequency with which tuberculosis has been met with in horses that had been so fed, becomes very striking." It is obvious that if we give milk to horses, either in health or disease, we should sterilise the milk in the event of our not being certain that the cows from which the milk was taken were free from tuberculosis. In sterilising milk by heat, we should be careful, if possible, not to allow it to attain a higher temperature than, say, 90° C. (taking the death-point of the tubercle bacilli at 85° C., see p. 154), so as to avoid imparting to the milk the peculiar taste which boiling gives it, and which is disagreeable, as a rule, to horses. Allowing it to get even in the slightest degree smoked, renders it still more unpleasant to the would-be equine drinker.—Tr.

chial and mediastinal glands are swollen up to the size of a man's fist, are hard to the touch, and have eminences on their surface. Their capsule is thickened and on section they appear grey with interspersed yellow foci. The portions of the pleuræ which cover the tubercles may also become thickened.

The serous membranes of the thorax and abdomen often show changes similar to those of bovine tuberculosis. The pleuræ are covered with single or conglomerated grey-yellow, grape-like tubercles, from the size of a poppy seed to that of a hemp seed, and sometimes with larger tubercles which may also become united into thick, nodulose, plate-like layers. Exactly the same changes are found on the perietal and visceral peritoneum, omentum, and mesentery, in the form of nodules varying in size from a millet seed to a walnut, partly isolated, partly strung together in grape-like or plate-like lumps, which may unite together and form tumours that may be as large as a man's fist. The mesenteric glands, like the bronchial glands, suffer change, and form tumours which may be as small as a walnut or as large as a man's fist, or may assume the appearance of a string of beads that may be as thick as a man's arm, and frequently weigh as much as 20 kilos. Similar changes are sometimes met with in the internal iliac glands. We also find tubercles of various sizes, and sometimes in great numbers, in the liver, in the spleen, which is often enormously enlarged, and in the kidneys, with simultaneous swelling of the portal, splenic and renal lymph glands. Tuberculosis of the bones (ribs and dorsal vertebræ), with disintegration of the bone tissue, formation of caseous foci in the bones, and periosteal proliferations have been observed. It is also stated that tubercular changes occur in the intestine.

**Symptoms.**—The aspect of equine tuberculosis is not very characteristic. Clinically, we may divide the disease into two principal forms, namely, a pectoral and a gastric. In the former, we usually find only symptoms of "broken wind" or asthma, such as chronic difficulty in breathing, cough, and emaciation, which in some horses progresses so rapidly that they become almost a skeleton in the course of from 6 to 12 months. In others, we find foci of dulness in the lungs, bronchial breathing, and râles. In one case, the coughing up of a necrotic, bacilli-laden piece of lung tissue was noticed. A case described by Schindelka reminds one vividly of human phthisis; the symptoms having been bronchitis, rapidly increasing debility,

loss of appetite, anæmia, variations in internal temperature, lingering pleuritis, harassing cough, excessive difficulty in breathing, great emaciation and, strange to say, polyuria. Nocard also observed severe polyuria and very irregular increase in temperature,  $1^{\circ}$  to  $1.5^{\circ}$  C. In one instance, he found tubercular deposits in the peritoneum by introducing his hand into the rectum.

The second form of tuberculosis is a grave intestinal affection, which is evidently the disease that was formerly described by Strauss, Träger and others under the name of umbilical pyæmia, except when the symptoms were identical with those of pyæmic polyarthritis. The descriptions of the disease correspond exactly with those of tuberculosis of the intestines and mesenteric glands (*Phthisis sive tabes mesenterica*). The foal gets out of health, becomes emaciated, tucked-up in the flanks, and pot-bellied; the coat becomes harsh and loses its naturally glossy appearance; and the animal suffers from digestive disturbances, such as constipation alternating with diarrhœa, and from slight attacks of colic. The foal becomes debilitated, prostrate, and finally dies from exhaustion or profuse diarrhœa. On making a *post mortem* examination, we find enlargement, caseation, and induration of the mesenteric and bronchial glands and catarrhal changes of the gastro-intestinal mucous membrane. Schortmann has described an interesting case of an adult horse suffering from umbilical pyæmia which could be traced to tuberculosis caused by feeding. In two cases, equine tuberculosis took the form of breast tumour (Röbert and Johne).

[McFadyean remarks on the frequency with which marked stiffness of the neck is a striking symptom of tuberculosis in the horse; and refers the cause of this stiffness to tuberculous ostitis and periostitis of the implicated cervical vertebræ.]

**Differential Diagnosis.**—An attempt to distinguish between equine tuberculosis and glanders may be made by means of the inoculation of tuberculin or mallein. We can also form an absolutely correct conclusion on this subject from an exact anatomical and bacteriological examination *post mortem*. The demonstration of tubercle bacilli by means of the Koch-Ehrlich staining method, or by one of those described on pages 180 *et seq.*, is decisive, as is also the result of the inoculation of guinea-pigs. Csokor points out that characteristic anatomical and histological differences exist between the nodule of glanders and tubercular new growths. He states that the nodules in the lungs in cases of equine tuberculosis are not so well defined as those of

glanders; that they possess a distinct inclination to form groups; and that each respective nodule becomes centrally caseated. He also remarks that the nodules (tubercles) in miliary tuberculosis of the lungs possess a fundamental substance which consists of three different kinds of cells, namely, giant cells, epithelioid cells, and round cells. The nodules of glanders, on the contrary, show only one layer of round cells, or connective tissue fibres, around a caseous, disintegrated centre.

#### CANINE TUBERCULOSIS.

**Occurrence.**—Although tuberculosis is not a very rare disease in dogs, it does not occur so frequently in them as in cattle and pigs. During the last few years, over 100 cases have been observed: 40 by Cadiot; 40 by Fröhner; 28 by Jensen; and 11 by A. Eben. Its frequency appears to vary greatly in different countries and towns. Among 70,000 dogs which, from 1886 to 1894, entered the Berlin clinic, 40, that is to say, 0·05 per cent., were affected. A similar number was found among the 9,000 canine patients at the Alford clinic during the years 1891 to 1893, thus giving a percentage of 0·44, which is about 9 times more than in Berlin. Out of 400 in Dresden, 11 (2·7 per cent.) were tuberculous. The percentage of tuberculosis in dogs was highest in Copenhagen.

**Pathogenesis.**—The reception of tubercle bacilli in dogs takes place most frequently by means of the lungs, which consequently are more often found (75 per cent. of all cases), *post mortem*, to be tubercular than other organs. Infection in them, as in man, is generally transmitted by inhaling tubercular dust in rooms, etc. Less frequently the tubercle bacilli are received through the digestive apparatus, in consequence of swallowing tubercular sputum or tubercular food. Absorption through the skin is rarest of all. It has been amply proved that when the dog becomes tuberculous, the infection has been derived, as a rule, from mankind; especially by inhaling the dust of a room occupied by a consumptive owner, licking up expectorated sputum, and eating food which has been masticated by a phthisical patient. On the other hand, the serious danger to human beings of having tuberculous dogs in their immediate neighbourhood should not be disregarded.

**Symptoms.**—As in human beings, canine tuberculosis runs

a chronic course usually in the form of pulmonary phthisis. In many cases, no conspicuous external symptom of the disease can be perceived, even for a very long time, on which account owners often remain unconscious of the fact during the life of the animal. The symptoms, however, vary greatly according to the site, extent, and age of the tubercular changes. Usually the disease assumes the aspect of chronic pneumonia, chronic pleuritis, hydrothorax, or chronic bronchial catarrh. The chief symptoms are : cough, dyspnœa, rapidly increasing emaciation, weakness, and a very irregular, atypical fever. Physical examination of the lungs shows dulness, the presence of cavities, rhonchi, suppressed respiratory murmurs, etc. Sputum is seldom observed, because the patient swallows it. Towards the end, there is accompanying diarrhœa and sometimes ascites. By that time the animal is reduced almost to a skeleton. Acute miliary tuberculosis seldom becomes developed.

**Anatomy.**—Canine pulmonary tuberculosis assumes many different anatomical aspects. Most frequently we find cavities and caseous foci which usually lead to rupture into the pleural sacs. In other cases, the *lungs* contain large pneumonic or small miliary tubercular foci. We find, comparatively often, chronic indurating broncho-pneumonia, with central softening ; or we may observe chronic interstitial indurating pneumonia. The lungs frequently adhere to the walls of the ribs and sometimes to the neighbouring lymph glands. Besides this, there may be present excessive compression of the lungs with atelectasis, carnification, œdema of the lungs, emphysema of the lungs, and bronchitis with bronchiectasis.

Tuberculosis of the *pleuræ* appears in various forms. The patient may suffer from either a serous or a sero-fibrinous pleuritis, with considerable discharge, into the thoracic cavity, of fluid, which, in some cases, assumes a hæmorrhagic character ; or, just as frequently, a dry, granular, callous, adhesive and contracting pleuritis, which leads to adhesions of the lungs. There may also be present a disseminated miliary, pleuro-tuberculosis. Emphysema and hydrothorax may be seen as secondary phenomena.

Tuberculosis causes the thoracic *lymph glands* (bronchial glands, anterior and posterior mediastinal glands, and in a few cases the inferior cervical glands) to become more or less enlarged and frequently to adhere to one another, so that they sometimes form sarcomatous conglomerations the size of a man's fist. The



tumours show on section a dirty white colour and are infiltrated with foci of softening or tubercles. The mediastinum often becomes thickened and swollen to an unrecognisable extent. In combination with the swollen lymph glands, it sometimes forms between the two lobes of the lung, sarcomatous, lardaceous tumours which are covered with fine proliferations and remind us of those of bovine tuberculosis. These tumours show on section, softened, caseated and calcareous foci.

The diseased condition of the *pericardium* manifests itself partly by a sero-hæmorrhagic, partly by a fibro-granular, adhesive pericarditis, and partly by miliary tuberculosis of the perietal and visceral portions of the pericardium. Hydropericardium is sometimes present.

Tuberculosis of the *peritoneum* is less frequent than that of the pleuræ; the changes being almost identical in both. Swollen proliferations, similar to those of the mediastinum, sometimes form on the omentum. We seldom find intestinal tuberculosis (intestinal ulcers with swelling of the mesenteric glands).

Tuberculosis of the *liver, spleen, testicles, kidneys, etc.*, is usually miliary.

**Statistics.**—The respective participation of individual organs in canine tuberculosis is shown by the following figures. According to Cadiot, the lungs were affected in 80 per cent. of all the cases; pleuræ, bronchial glands, mediastinal glands and liver, in 60 per cent.; kidneys, in 45; peritoneum and omentum, in 30; pericardium and mesenteric glands, in 25; spleen and intestinal mucous membrane, in 10; and the heart, in 5 per cent. Exudative pleuritis and ascites were present in 50 per cent.; and exudative pericarditis in 12 per cent. General tuberculosis was met with in more than one-third of all the affected dogs. Jensen states that the lungs were tubercular in 75 per cent. of all the cases. In about half the number, the bronchial glands, mediastinal glands, and serous membranes, especially the pleuræ, were involved. The liver and kidneys were equally often affected. Eber found that the lungs were implicated in 80 per cent. of the cases; the bronchial glands, in 70 per cent.; the pleuræ, in 35; the pericardium, in 10; and general tuberculosis was present in 10 per cent. of the cases. According to Fröhner, the percentage for the lungs including the pleuræ, was 90; for the liver, 80; for the bronchial and mediastinal glands, 50; for the pericardium, 40; for the kidneys, 25; and for the spleen, and for cases of general tuberculosis, 10 each.

**Diagnosis.**—The history of the case in dogs, often leads us to suspect the presence of tuberculosis. We usually learn that the patient has suffered for a considerable time from cough, dyspnœa and increasing emaciation, and that every means of alleviation which had been tried had failed. The phthisical

appearance of the owner may lead us to suspect tuberculosis in the dog. Generally, a diagnosis of tuberculosis will be justified, if the dog is suffering from chronic bronchitis, pneumonia or pleuritis, and from great emaciation. An exact diagnosis is, however, possible only by bacteriological examination, inoculation, and, to some extent, by the use of tuberculin. We may find bacteriologically the tubercle bacilli in few numbers in the sputum and in the discharge from the nose (Fröhner and Bang); but can obtain more reliable results by examining the pleuritic exudate obtained by puncture of the thorax and by injecting it intraperitoneally into guinea-pigs (Cadiot).

Tuberculin (0.05 gramme for large dogs) is in many cases a valuable auxiliary diagnostic agent with dogs. It is a surprising fact that the reaction sometimes takes place as quickly as from 2 to 5 hours after the inoculation. It is, however, unreliable in cases of advanced and general tuberculosis and with feverish animals, and in such cases it is not altogether without danger; for it not unfrequently causes exacerbations, lowering of the internal temperature, and may even set up fatal complications. In some instances, non-tuberculous dogs react to tuberculin.

**Therapeutics.**—Advanced tuberculosis of the dog is incurable; for which reason and on account of the danger of infection to man, tuberculous dogs should be destroyed without delay. In the early stages of the disease, we may try the effect of strengthening diet, open air, hygiene, and certain medical agents, such as creosote and creolin. In one case, we obtained considerable improvement by continued inhalations of creolin.

#### TUBERCULOSIS OF THE CAT.

Tuberculosis is not very rare in cats. As a rule, the source of infection, as in dogs, is obtained from a tuberculous owner. Cats in large towns are especially liable to become affected. In Berlin, out of 100 sick cats, one was tuberculous (Fröhner). Jensen collected 25 cases in Copenhagen. The chief symptoms, as in dogs, are: excessive emaciation, anæmia, debility, dyspnœa, violent coughing, and dulness of the lungs on percussion. *Post mortem* we find the lungs and the abdominal organs most frequently implicated. Jensen observed that the lungs are very often affected; but the pleuræ, exceedingly seldom. The mesenteric glands and kidneys suffer in many cases; but the intestinal canal and liver very rarely. The

milk-white colour (from fatty degeneration) of the section surfaces of the larger tubercles is very striking. The lymph glands of the head and neck are frequently attacked. In two cases, the uterus and testicles respectively were tubercular; for which reason Jensen concludes that infection through coition has been absolutely proved in cats. In a few cases, tuberculosis with secondary affection of the axillary glands was found in the subcutaneous tissue, as a result of wounds. One cat suffered from arthritic tuberculosis. Jensen states that cats are affected chiefly through the digestive canal—a fact which we may infer from the frequency of the disease in the mesenteric glands. Not uncommonly, the digestive organs only are affected; although it appears that infection through the lungs is not rare. Nocard observed tuberculous arthritis in a cat which had been fed experimentally with milk that contained tubercle bacilli. Bollinger has described two cases of miliary tuberculosis, in which tubercles were present in the pancreas. In one case, Zschokke suspected that infection had been transmitted by man.

#### TUBERCULOSIS OF SHEEP.

Tuberculosis seems to be somewhat rare among sheep. Only 5 cases were found among 340,000 sheep killed in the Berlin abattoir in the course of one year, 1888–1889. Among 130,000 sheep killed in 1894 in Saxony, there was a tuberculous percentage of 0.15. The anatomical changes in ovine tuberculosis closely resemble those of bovine tuberculosis. Several observers suspect that infection is transmitted by tuberculous cattle through cohabitation and milk. Great emaciation, anæmia, and cough are the chief symptoms. In a case described by Rasmussen, a lean six-year-old sheep, slaughtered in the Copenhagen abattoir, showed a number of defined tubercles, varying in size from a hemp seed to a hazel-nut. These tubercles partly projected out of the surface and were partly imbedded in the tissues. They all consisted of a thick capsule of connective tissue and caseous contents. The bronchial, mediastinal and thoracic glands and the glands under the shoulder blade were considerably swollen, firm, knotty, and studded with a calcareous deposit. Some isolated pedunculated new growths were on the pleuræ, and the liver was partly covered with tubercles and partly infiltrated. The first lumbar vertebra contained a cavity the size of a nut, and was filled with mortar-like caseous material. Similar processes were found in the two last thoracic vertebræ.

## TUBERCULOSIS OF GOATS.

Goats have been considered by many to be refractory to tuberculosis. Acting on this erroneous supposition, goats' milk has been recommended as a substitute for cows' milk, in order to guard the consumer against tuberculosis. In fact, there have been established cure-institutions in which goats' milk was exclusively used. This misconception arose from the fact that formerly tuberculosis was seldom found in goats; because *post mortem* examinations of these animals were very rarely made in abattoirs. Veterinary literature proves that this disease is not at all uncommon in goats. In Saxony 0.6 per cent. of the 1,500 goats killed in 1894 were found to be tuberculous. It seems that, as a rule, tuberculosis in goats is general. In a case reported by Sluys and Korevaar, a goat which had been brought up on cows' milk showed all the signs of pulmonary phthisis; and general tuberculosis, starting from the intestinal tract, was found *post mortem* in this goat. At the Munich abattoir, Magin observed in a goat that almost all the internal organs, even the spongy tissue of the vertebræ, were infiltrated with tubercles so that the flesh had to be absolutely forbidden to be used as food. In spite of the fact that the animal was well nourished, numerous dirty-white tubercles of a caseous character, varying in size from a millet seed to a pea, were met with in the parenchyma of the lungs, liver, and spleen, and in the pleuræ and peritoneum; and the mediastinal glands were swollen and infiltrated with tubercles. Edgar found in a five-year-old goat the entire lung infiltrated with small, greyish-yellow nodules. Eichhorn inoculated 28 goats out of a herd of goats in which a case of tuberculosis had occurred, with from 0.02 to 0.05 gramme each of tuberculin; the result being that, with a single exception, all the animals showed an increase of temperature of from 0.6 to 2.8 C.; and eighteen of them, a rise of over 1° C. Three of these animals which were slaughtered, proved to be tuberculous. In all probability, the majority of the remainder was affected.

## TUBERCULOSIS IN WILD MAMMALIA.

The majority of the wild animals exhibited in zoological gardens, such as monkeys, apes, lions, tigers, panthers, giraffes, etc., fall victims to tuberculosis.

## TUBERCULOSIS OF BIRDS.

**Occurrence.**—Tuberculosis is one of the most common diseases of birds. Amongst 1,100 hens dissected at the Leipsic

veterinary clinic, 106, namely, 10 per cent., were found to be tuberculous. Among the 700 parrots treated, from 1886 to 1894, in the Berlin clinic, 170 (25 per cent.) were similarly affected. This disease attacks all kinds of birds (hens, pigeons, pheasants, peacocks, parrots, etc.), and not unfrequently occurs as an enzootic. In poultry, tuberculosis is generally located in the intestines, which, consequently, is the usual entrance of infection. The transmission takes place most frequently by means of the fæces of affected fowls, which fæces contain great numbers of bacilli. Not uncommonly the infection is derived from tuberculous human beings by the eating of tubercular sputum and of food which had been previously masticated. It also appears that the disease is transmitted by the milk and flesh of tuberculous cattle. The heredity of tuberculosis, which is believed in by poultry breeders, has been proved by the experimental investigations of Maffucci.

Recently doubts have been expressed about the identity of the tuberculosis of birds with that of mammals, including man. Koch considers that the bacilli of avian tuberculosis differs from true tubercle bacilli, although it is closely related to them. He leaves open the question of their having the same pathogenic effect on man or not. Rivolta, Maffucci, Baumgarten, Strauss, and Gamaleïa have also pronounced in favour of making a distinction between the two diseases and between the two bacilli; because human tuberculosis can be very easily transmitted by inoculation to guinea-pigs, which are almost immune from the tuberculosis of birds. In contrast to human tuberculosis, the tuberculosis of fowl can be very easily transmitted to fowl and rabbits. The more recent investigations of Cadiot, Gilbert, Roger, Fischl, Courmont, and others, tend to show that these two forms of tuberculosis are produced by the same species of bacilli, although by different varieties; the differences between them having been produced by differences in their respective modes of nutrition.

**Anatomy.**—In poultry, the anatomical changes are chiefly found in the liver, spleen, and intestines. The liver is infiltrated, especially on its surface, by tubercles, varying in size from a millet seed to a pea, or even a walnut. These tubercles frequently become massed together, and show, according to their age, a white, grey, or yellow colour. In the centre they are caseous or calcareous, and contain great numbers of tubercle bacilli. Similar changes are found in the spleen. On the in-

testinal mucous membrane we may see miliary tubercles which are about the size of a pea, and which subsequently ulcerate. Tubercles also form on the peritoneum, mesentery, kidneys, ovaries, etc. Tuberculosis of the lungs, guttural pouches, heart, and pericardium is comparatively rare. The disease pretty frequently invades the lymph glands and articulations, especially those of the wings and feet, in the neighbourhood of which large swellings and abscesses with caseous contents become developed, thus causing tubercular arthritis and peri arthritis. Tubercles and ulcers also form in the bones, skin, and subcutaneous cellular tissue.

**Symptoms.**—The symptoms of tuberculosis of birds are not very characteristic. The sufferers are feeble and become gradually emaciated, often almost to a skeleton. The comb and wattles are pale and dried up. The mucous membranes of the eyes and mouth are pale, and the birds suffer in particular from chronic disturbances of digestion, such as loss of appetite, vomiting, and diarrhoea. Towards the end, great debility and paralysis supervene. The diagnosis becomes somewhat easier when these symptoms are associated with local tuberculosis in the articulations, bones, and skin, in the form of swelling of the joints and bones, and of tumours and unhealthy ulcers in the skin. The disease usually runs a rather slow course.

#### TUBERCULOSIS OF PARROTS.

This form differs in some points from that of domestic fowl. The infection usually takes place through the respiratory apparatus on account of the birds inhaling the bacilli of human sputum along with dust deposited in dwelling-rooms. Next comes infection through the digestive apparatus by eating food which had been masticated and then rejected by the owner of the parrot. The skin, especially that of the head, and the mucous membrane of the eyes also form, at times, the port of entrance. Tuberculosis of the skin is characterised by new growths of horny skin, which frequently attain a considerable size. They can be easily broken off, and contain numerous tubercle bacilli in their basal granulation tissue. Tuberculosis of the lungs and conjunctiva is often met with. According to a compilation by Eberlein, the skin was infected in 52, the tongue in 16, and the bones and articulations in 12 per cent. of all the cases. The tubercles of the skin vary in size from the head of a pin to a hen's egg; are spherical or oval and semi-solid; and have a horny, scaly surface. From a diagnostic point of view, it is important to note that the tuberculosis of parrots is distinguished by the presence of an enormous number of bacilli. Eberlein found numerous bacilli, on cutting and making punctures into the new growths, and by examining the fæces, nasal discharge, and saliva. The treatment of tubercular new growths belongs to the domain of surgery. Relapses are common.

## PSEUDO-TUBERCULOSIS.

This name is given to some diseases which closely resemble tuberculosis, anatomically and clinically, but are not caused by the bacillus tuberculosis. These diseases are of diagnostic importance from the fact that they occur in cattle, and in animals which are used for inoculation experiments, in both of which cases they may become confused with true tuberculosis. A correct conclusion can be arrived at only by bacteriological means.

1. Kitt has described in *cattle* a bacillar, caseous, broncho-pneumonia which could not be clinically distinguished from tuberculosis. On bacteriological examination he found bacilli of from 1 to  $1\frac{1}{2}$   $\mu$  long, of about the thickness of the bacilli of erysipelas and capable of being stained with Gram's solution; but no tubercle bacilli. A similar disease has been described as "bovine glanders," by the French veterinary surgeon Nocard. Courmont observed a peculiar bacillus in tubercular-like pleural nodules of cattle. Stöhr noticed in sucking calves a caseous non-tubercular pneumonia with a singular bacillus. We may draw attention to the pseudo-tubercular pneumonic foci which are met with in diseases caused by pulmonary worms (strongyli) in cattle and sheep, such as parasitic tuberculosis due to nematodes or "nematode tuberculosis." Hink has observed in the lungs of a large ruminant, tubercle-like formations caused by "*aspergillus fumigatus*." Preisz and Guinard found small short bacteria with rounded ends in the tubercle-like calcareous nodules of the kidneys of a sheep.

2. In *guinea-pigs* and *rabbits* we meet with various processes which resemble tuberculosis. Eberth, Dor, Parietti, Zagari, Chantemesse, Charrin and Roger have described affections closely resembling either tuberculosis or glanders in these two very important experimental animals. These simulating diseases could be distinguished from the true ones only by their respective bacteriological differences. Pfeiffer described in detail a specific and pathogenic *bacillus pseudo-tuberculosis*, which he considers to be the cause of the so-called pseudo-tuberculosis of guinea-pigs and rabbits. This bacillus can be stained by Löffler's solution but not by that of Gram. It has many points of resemblance to the bacillus of glanders, but differs essentially from that of tuberculosis. Inoculations of it produce caseous nodules in the liver and spleen, varying in size from a pin's head to a pea, and caseous foci in the lungs resembling the growths of glanders. The fact that the abdominal organs participate in the disease to a far greater extent than the lungs, serves to macroscopically distinguish pseudo-tuberculosis from true tuberculosis. The pseudo-tubercular nodules contain no giant cells, and only a few epithelioid cells. The inoculated animals die quicker than those inoculated with true tuberculosis, on which account the nodules never undergo dry caseation, but show in their centre a pus-like fluid, as in glanders.

3. Ebstein and Nicolaïer have found in the kidneys and lungs of *dogs* and *cats* a zooparasitic tuberculosis, which appears in the form of nodules, and which is caused by cylindrically-shaped worms. According to Laulanié, similar nodules are produced in the lungs by *strongylus vasorum*. Potain has described in pigeons, tubercle-like formations caused by *aspergillus*, and Cornil has reported the presence, in an antelope, of similar affections caused by an oval bacterium.

## ACTINOMYCOSIS.

Actinomycosis in general—Actinomycosis in Pigs, Horses and Sheep—Actinomyces Musculorum suis—Botryomycosis in Horses—Actinomycosis of Man.

**Etiology.**—Actinomycosis is due to ray fungi (actinomyces), which belong to the pleomorphic bacteria and to the cladothrix group of fission algæ, or fission fungi (Boström). According to recent investigations, they appear to belong to the family of streptothrix and to occur in different varieties. The characteristic feature of these fungi, as compared to other bacteria, is that they grow, not as single individuals, but as regularly built-up, combined structures. These colonies or granules measure  $\frac{1}{5}$  to  $\frac{1}{2}$  mm. in diameter, and consequently may be seen by the naked eye as spherical bodies which are about the size of grains of sand. According to their age, they are grey and gelatinous, and resemble small lumps of mucus; or they are greyish-white, opaque, and sometimes yellow, brown, or even green. We can distinguish in the radially arranged colonies, two layers, namely, a peripheral layer of clubs, and a central layer of threads. The former consists of pear-shaped bodies, the so-called clubs, in the axis of which runs a thread of mycelium; the contents breaking up into spore-like granules. When these clubs open out at their apex, after the manner of a bud, secondary clubs become developed, in the form of finger-like or hand-like processes. As demonstrated by Boström, these clubs are not organs of fructification (gonodia), but are forms of gelatinous degeneration. The central thread layer consists of an entangled plexus of filaments formed by rods, which are of various lengths and are motile in water, and by long spiral threads. Moreover, we find a granular substance consisting of round cocci-like structures (spores) which have sprung from the filaments. The true organs of fertilisation are found in the interior of the actinomyces-granules, in the germ stratum of the filamentous layer. The growth of new colonies takes place by the separation of the filaments or spores of the old granules. It is probable that the germs of the fungus in the form of rods or filaments are carried away by the leucocytes (phagocytosis and chemiotaxis). The tissues lying in the neighbourhood of the tufts die and become surrounded by an outer zone of leucocytes, and later on by granulation tissue. The gelatinous degenera-



tion of the nodules (formation of clubs) is accelerated by increase of granulation tissue. The tufts frequently become calcareous.

**Bacteriology.**—Pure cultures of actinomyces on dried blood serum, which are best prepared by previously rubbing down the granules, form at first, according to Boström, a thin gelatinous layer, consisting of fine transparent filaments. The layer grows and looks as if it were sprinkled over with chalk in consequence of the accumulation of little dots containing numerous cocci and filaments. After a fortnight, these white dots become pale yellow, pure red, or brick red in the centre, and confluent ; whilst on the periphery a transparent dirty-white stratum forms. Old cultures become puckered and hard. The fungus grows in a similar way on agar-agar, glycerine agar, and gelatine. On potatoes it forms grey, yellowish, and finally white granules. The growth is not stopped by the exclusion of air (facultative anærobic bacteria). Experiments with cultures in animals have been mostly negative. Only Wolff and Israël have succeeded in producing actinomycosis by inoculation.

**Pathogenesis.**—Actinomyces, which are found originally on plants, seem capable of entering the body in various ways ; although the digestive canal is usually their first point of attack. According to Brazzola, they vegetate chiefly on *hordeum murinum*. He discovered quantities of them between the vegetable fibres of broken particles of barley which were imbedded in the gums. John, Piana, Boström, and others have found this fungus abundantly among the awns of corn which were in the tonsils of pigs and in the tongues of cattle. It is generally supposed that transmission takes place by the fungus settling in small wounds on the buccal membrane, in the ducts of the glands, and perhaps in the alveoli of diseased teeth, or during the shedding of the milk teeth ; and that it grows and develops from these sites. It seems, especially with cattle, that the awns of grain, covered with this fungus, penetrate between the teeth and into the gums and tongue. If they have once effected a lodgment, they can be removed only with difficulty on account of the peculiar position of the hair on the beard, just as we see with spikes of rye which have become stuck on the sleeve of a coat. A favourite site for actinomycosis of the tongue is a point on the upper surface of the tongue midway between the dorsum and the tip. Hentschel and Falk point out that about 9 per cent. of affected cattle showed on that spot, lesions of epithelium which served as starting points for the infection. Boström believes that the ray fungus develops exclusively on grain, particularly on the awns of barley ; and that actinomycosis is caused only by the penetration of such infected parts of plants. We find in dry grain, symmetrically-arranged air spaces, which have

fissured openings in which the fungus may remain dormant for more than a year in a dry condition. After that, when it is transferred with the awn to animal tissue, it begins to grow luxuriantly. It appears, as a rule, that cattle become infected only when they are fed on dry food (Imminger and Claus). The immigration may however begin in the lungs by fungi contained in the inhaled air ; in wounds of the skin ; and by the openings of the ducts of the teats. Gooch observed that in a large herd of cattle which had been treated by setons, the majority of the wounds showed actinomycosis. A general infection over the whole body by means of the blood, as in tuberculosis, seems possible, although it could rarely occur. With regard to the rapidity of growth of the actinomycomata, Andersen observed that tumours of greater or less size developed in 17 cows out of 20 which were out on grass during the 5 months from July to December.

Some observations suggest the idea that *swampy districts* are favourable to the development of actinomycosis. Bang and Jensen remarked on the epizootic distribution of the disease among the cattle of a seaside district of Denmark, in which place the sea had receded from a portion of a bay and left a lake on the reclaimed land, which was exposed to inundations. Cattle which had been fed on barley grown on the reclaimed ground of this inundated district acquired the disease. Imminger, Preusse, Claus, Roger, Davaine, and others, have made similar observations on the influence of swampy and inundated pastures.

*Actinomycosis is not a contagious disease.* Attempts to transmit to other animals have been made by various persons, but always without result (Rivolta, Bollinger, Siedamgrotzky, Peroncito, Johne, Ullmann, Bodemer, and Boström). Cattle, calves, goats, sheep, pigs, dogs, cats, rabbits, and guinea-pigs have remained healthy after inoculation. Only in a few cases, reaction to inoculation was displayed by the formation of inflammatory demarcation granulations. The asserted positive results obtained by Ponfick, Israël, Rotter, and Hanau are, according to Boström, simply residual and encapsuled inoculation nodules. It seems that the ray fungus has a pathogenic effect only in the stage of development connected with the awns of grain, and that it loses its power of transmission as soon as it has entered the animal body, on account of undergoing some form of involution (calcification, etc.). The negative result of the inoculation experiments is of great importance for elucidating the question of the transmissibility of actinomycosis from one animal to another,

or from one of the lower animals to man. The foregoing considerations tend to prove that infection cannot take place in this manner.

**Occurrence.**—Actinomycosis has been observed in cattle, pigs, horses, sheep, and man. The ray fungus was first seen about 1860 by Perroncito and Rivolta, and by Hahn in 1870. Bollinger furnished the first exact description of the disease in 1877. Harz was the first to examine the fungus botanically and to give it the name actinomycosis (ray fungus).

Actinomycosis is generally sporadic, though sometimes enzootic. Preusse has described an epizootic outbreak of this disease in West Prussia, by which 20 per cent. of all the cattle became infected. According to Imminger, the disease is particularly rife in the Upper Palatinate and neighbouring parts of Upper Franconia. He is accustomed to treat yearly more than 100 cases of bovine actinomycosis in the Bavarian Upper Palatinate. Claus has collected 105 cases. Actinomycosis appears to be very common in Russia. Of all the cattle slaughtered in Moscow, from 2.5 to 5.5 per cent. are found to be infected with it (Oskolkow). Barret states that Canada shows the comparatively high percentage of 2; Salmon puts the percentage for the remainder of North America down at 0.2. The disease is somewhat rare in France (Cadiot). The percentage in La Vilette was 0.7 (Nocard). Peletti has seen hundreds of cases of actinomycosis in Italy. His experience of the disease impressed upon him the fact that it is especially liable to occur after an epizootic outbreak of aphthæ, on which account he connects the presence of ulcers in the buccal membrane with the pathogenesis of actinomycosis. Neuwirth has made a similar observation. In Denmark the outer soft parts of the face and the neighbouring cervical region are most frequently attacked (Bang). In England the usual seat of the disease is the tongue; and in Russia, the lips.

The statistics of the abattoirs give the following figures. In Berlin there were 21 cases from 1885 to 1886 among 100,000 cattle (1 in 5,000); and 2 out of 300,000 pigs (1 in 150,000). In Augsburg, 8 in 23,000 cattle (1 in 3,000). In Bremen, 2 among 8,500 cattle (1 in 4,520), and 3 out of 25,000 pigs (1 in 8,000). In Stuttgart, 12 among 12,000 cattle (1 in 1,000); and in Hanover, 1 in 10,000 cattle. Ivanow found 2,000 cases of actinomycosis during 2 years in the abattoir of Moscow, in the slaughter-house of which Mari observed 540 infected carcasses

out of 150,000 (1 in 3,000) cattle that were killed. At Warsaw there were 70 cases out of 350,000 beasts (1 in 5,000).

**Anatomical Changes.**—Boström states that actinomycosis is a specific inflammation which occurs in the three following degrees in our domestic animals: (1) A degenerative granular inflammation (tongue); (2) a progressive purulent granular inflammation (cold abscesses); (3) a fungous actinomycoma (pharynx and skin). First of all a granulation tumour develops in the neighbourhood of the actinomycotic granules, in consequence of inflammatory reaction. This tumour leads to the formation of tubercle-like nodules and large rounded or lobulated tumours, which are termed actinomycomata by Johne. These actinomycomata are sometimes very soft, of a sarcomatous consistence, and of an orange colour. At other times they are rather hard and firm to the touch, of the consistence of a fibroma, of a whitish-grey colour, and sometimes spongy. They consist of a connective tissue stroma in which numerous nodules the size of a millet seed up to that of a pea are interspersed, and shine through the surface. These nodules possess the microscopic structure of granulation tumours and contain the sulphur-yellow actinomycosis granules, which are about the size of a grain of sand. They may form larger nodules by becoming massed together. Purulent disintegration of the actinomycomata gives rise to "cold abscesses," which are surrounded by very soft granulation tissue, and contain the small yellow tufts of the fungus.

Actinomycosis of cattle usually appears in the upper or lower *maxillary bones*, where it generally produces very large tumours in their soft tissues and osseous tissue. These tumours were formerly known under the name of "wens," *spina ventosa*, sarcoma of the jaw, cancer, etc. Actinomycosis of the jaw commences with flat granulations on the gums and mucous membrane in the neighbourhood of the teeth, and spreads to the periosteum, and finally to the medullary tissue of the bones, where it soon gives rise to an osseous tumour. This swelling is the product of an ossifying periostitis, and of a rarefying granular osteitis, which gives rise to the spongy cavities, or *lacunæ* that are highly characteristic of macerated bone, and are due to atrophy of the osseous trabeculæ. On the other hand, the simultaneous periostitis is followed by an increase of the circumference of the bone with a peripheral osseous deposit. From the maxillary bone, the actinomycotic granulation substance may

advance, either to the skin or to the oral cavity, in the direction of the molar teeth, which become displaced, or finally become affected by the morbid process in the same manner as the bone.

In cases of actinomycosis, the disease very frequently attacks the *tongue*, in which it takes essentially the form of an indurating glossitis (actinomycotic sclerosis or macroglossia), on which account it was formerly called "wooden tongue" (*Holzzunge*, and *langue de bois*). We notice, first of all, underneath and on the sides of the mucous membrane of the tongue, circular, somewhat raised brown spots, through which shine very minute yellow nodules. Later on, we find similar spots, varying in size from a grain of millet to a pea, in and under the mucous membrane and in the intramuscular connective tissue. These actinomycotic nodules are round, hard to the touch, fibrous, and show a purulent, caseous, chalky or mortar-like substance in their centre. In the neighbourhood of these nodules there is a considerable proliferation of connective tissue, which leads to atrophy of the muscle fibres, or to induration of the organ ("wooden tongue"). On cutting through the tissues of the greatly enlarged tongue, we find it lardaceous, hard, and even gritty. The cicatricial shrinking of the proliferated connective tissue eventually causes contractions, erosions from friction, and various deformities of the tongue. The lymph glands of the tongue become studded with nodules and abscesses.

As a rule, the actinomycotic proliferations in the *pharynx* take the form of soft polypoid or fungoid nodules or lumps which have a short peduncle and smooth surface, and which vary in size from a pea to a goose's egg. These actinomycomata may give rise to difficulty in swallowing and attacks of dyspnoea. Similar nodules which sometimes have a broad base, and which vary in number, size, and consistence, may form in the *œsophagus*, *reticulum*, *larynx*, and *trachea*. Actinomycotic processes are of rarer occurrence in the *omasum*, *abomasum*, *intestines*, *peritoneum*, *omentum*, and *mesenteric glands*.

Actinomycomata of the *skin* and *subcutis* are found chiefly on the head and neck. They usually form elastic, firm nodules, the size of a hazel-nut up to that of a man's fist, or may be even larger. Sometimes these nodules are pedunculated; at other times they are attached to the skin by means of a broad base. They may present the form of a soft, granular, fungoid proliferation of the colour of red meat, and are then covered with brown crusts, or with a purulent secretion. At other times, yellow nodules, the size of a pin's head, shine through the surface of

these proliferations, in the neighbourhood of which, the skin becomes thickened and indurated. Cutaneous actinomycomata may attain a very large size. Actinomycosis of the skin may be primary or secondary. In the latter case, it takes its starting point from deep lying primary foci, in, for instance, the jaw, udder, or glands. Tumours and ulcerous erosions form on the mucous membranes, chiefly of the mouth and nostrils.

In many cases, the *lymph glands* in the neighbourhood of the larynx and the pharynx also become secondarily affected with actinomycosis from the mouth, larynx and pharynx. Actinomycomata are found most frequently in the subparotid glands (upper cervical glands). They form firm, round, oval, or even elongated nodules, the size of a walnut up to that of a man's fist, and contain, as may be seen under the microscope, numerous typical tufts. The submaxillary and parotid glands sometimes become affected.

Actinomycosis of the *lungs* occurs in two forms : First, that of disseminated, firm, whitey-yellow nodules which become calcareous in their centre and vary in size from a millet seed to a pea (miliary actinomycosis) ; second, that of larger purulent foci of softening (cavities), which may attain the size of a man's fist, and which are filled with a grey, muco-purulent fluid (lobular actinomycotic inhalation-pneumonia). The actinomycomata of the lungs may spread to the pleuræ, and even reach the surface of the body by penetrating through the ribs or intercostal muscles. The bronchial glands and the mucous membrane of the air passages of the head may become affected.

Actinomycotic processes may form on the *udder* (mastitis actinomycotica purulenta fibrosa), *spermatic cord* of castrated animals, *brain*, *spleen*, *liver*, *muscles*, *diaphragm*, *peritoneum*, *inguinal glands*, *vagina*, *uterus*, in and between the *cervical vertebrae*, etc. Actinomycosis, like tuberculosis, may become general.

**Statistics.**—The maxillary bones, tongue, pharynx, parotid glands and skin are the most frequent seats of the disease ; although exceptions to this rule are met with. Among the 105 cases collected by Claus, the maxillary bones (the lower jaw in the majority of such cases) were affected in 51 per cent. ; the tongue, in 29 ; the pharynx, in 7 ; the larynx and trachea, in 6 ; and the lungs, abdominal viscera and bones of the head, only in a few instances. According to Imminger, the head and neck are implicated in from 85 to 90 per cent. of the cases, and the tongue in only 4 to 8 per cent. Rasmussen found the jaw affected in 14 out of 15 cases in oxen. Among 201 diseased cattle observed by Kuritzin, there were only 3 maxillary cases ; the tongue being almost exclusively attacked. Actinomycosis of the tongue appears to be particularly

rare in France, in which country out of 130,000 cattle which showed 0.7 per cent. of actinomycosis, that organ suffered only in one instance. Oskolkow states that in Moscow the lips were involved in 50 per cent. of the cases. Mari gives the following figures from 541 cases of bovine actinomycosis which he collected:—

Skin . . . . .	271 cases
Submaxillary glands . . . . .	177 "
Bones of the head . . . . .	117 "
Lymph glands under base of skull . . . . .	51 "
Upper bronchial glands . . . . .	38 "
Lungs . . . . .	29 "
Inferior bronchial glands . . . . .	5 "
Tongue . . . . .	5 "
Pharynx . . . . .	4 "
Bronchial glands . . . . .	4 "
Peritoneum . . . . .	3 "
Pelvis . . . . .	2 "
Pleuræ . . . . .	1 "
Inguinal glands . . . . .	1 "
Wall of the thoracic cavity . . . . .	1 "
Wall of abdominal cavity . . . . .	1 "
Mediastinal glands . . . . .	1 "
Liver . . . . .	1 "

**Symptoms.**—Although actinomycosis is of greater interest to surgery and pathological anatomy than to internal medicine, it sometimes presents symptoms which belong to the domain of special pathology. Thus, in actinomycosis of the tongue, prehension of food, and especially mastication, are impeded and even rendered almost impossible; the tongue is swollen and painful to the touch; and copious salivation is frequently present. Difficulty in swallowing arises from implication of the pharynx; dyspncea, from new growths in the larynx; and chronic tympanites, from actinomycosis of the mediastinal lymph glands. In cases of actinomycosis of the outer surface of the neck, we find the parotid region swollen and covered with tumours of varying size. Actinomycosis of the cervical vertebræ may cause spinal paralysis as a consequence of atrophy of the spinal medulla from continued pressure. Actinomycosis of the lungs may also present the appearance of a chronic affection of the lungs (phthisis). The course of the disease is always of long duration. Cases of spontaneous recovery may take place, as in tuberculosis, in consequence of the growth becoming encysted and calcareous. Recent experiences with iodine have rendered prognosis more favourable. Actinomycomata of the skin, glands, and maxillæ can be treated surgically.

**Differential Diagnosis.**—The diseases which are most easily mistaken for actinomycosis are tuberculosis (tuberculosis of the lungs and lymph glands), bovine pleuro-pneumonia, and foot and mouth disease. It may be mistaken for simple glossitis showing new growths in the pharynx (polypus, fibromata, and sarcomata), sarcomata of the jaw, lymphomata, cysticercus mediocannelatus in the tongue, parotitis, cellulitis, etc. On this account, the diagnosis of actinomycosis during the life of the patient is by no means easy. Often the disease can be recognised only by a microscopical examination after slaughter. For diagnosis *intra vitam*, it is of importance to extirpate or to incise nodules or abscesses in or under the skin, and to submit them to an exact microscopical examination. The actinomycotic erosions which occur on the mucous membrane of the mouth differ from aphthous ulcers, by reason of their base being hard and leathery. In all cases, the diagnosis can be confirmed only by microscopical proof of the presence of the actinomyces tufts.

Simple microscopical examination with the addition of one drop of concentrated liquor potassæ suffices to demonstrate the presence of the ray fungus. The staining of the fungi gives a great deal of trouble and is not absolutely necessary. Among different colouring materials which have been used, we may mention a double staining with orseille and gentian violet, orcein, picro-carmin, hæmatoxylin, eosine and a mixture of aniline dyes. The cover glass preparations are made in the ordinary way. A small quantity of the pus or other material which is to be examined, is spread in as thin a film as possible on a cover glass. It is dried in the air and then passed, with the film side upwards, three times through a spirit flame, by which it must not be heated too highly. The cover glass preparation is then placed for two or three minutes in a solution of picrocarmin, or other colouring agent, after which it is cautiously rinsed in water or alcohol and examined in water, glycerine, or Canada balsam. If the ray fungi be present, they will show a bright yellow colour and the tissue will manifest a red stain.

**Therapeutics.**—Formerly the treatment of actinomycosis was exclusively surgical, as for instance, by extirpation, caustics, actual cautery, incision, parenchymatous injection, and painting, which purely operative methods are even now applied to actinomycomata that can be reached externally. The deeper lying tumours, especially actinomycomata of the pharynx and the larynx, cannot, as a rule, be treated by an operation. For these



surgically inaccessible tumours, the internal administration of iodide of potassium has been found to be a specific remedy. Thomassen, who in 1885 was the first to recommend internal treatment with iodine, advises that each of the bovine sufferers should get daily, for 14 days, 6 grammes of iodide of potassium \* dissolved in half-a-pint of water. The dose may be reduced to 4 or 5 grammes when convalescence sets in. It is stated that a visible improvement is noticeable after 8 days of this treatment ; and that the affection becomes cured, on an average in a fortnight, if the disease be restricted to the tongue and neighbouring soft parts. Numerous trials have proved, with a few exceptions, the specific curative effect of iodide of potassium (Fürthmeyer, Bass, De Jong, Nocard, Oster-tag, Deslex, Reeks, Perinni, Havas, Gooch, Soucail, Hohenleitner, Krug, Schwäbel, Walther, Salmon, Bang, and others). The internal treatment with iodide of potassium may be supplemented by external application of iodine in the form of the tincture, or of Lugol's solution, either of which can be applied by means of a brush or injected subcutaneously.

**Inspection of Meat.**—As we have already mentioned

\* I am inclined to think that non-success in the treatment of actinomycosis by the internal administration of iodide of potassium has, in many cases, been due to a too sparing use of the drug. In one instance of a three-year-old thoroughbred colt which was suffering from actinomycosis of the tongue, and which had derived no benefit from a course of daily doses of half an ounce, I obtained a perfect cure by at first quadrupling the dose and giving during 38 days 52 ounces. When I first saw the colt, his tongue was so greatly swollen, and was so hard and painful from the actinomycotic changes in it, that he was unable to eat any kind of forage, and was consequently in the last stage of starvation. After 10 days, during which time I kept him alive by a plentiful supply of raw eggs, he was able to eat grass fairly well. The course of recovery was marked by the tongue becoming smaller, softer, and more mobile ; by disappearance of the previous foetid smell from the mouth ; and by loosening of the actinomycotic nodules in the submucosa of the tongue, so that they could be readily shelled out with the finger-nail. On two occasions during this treatment, I discontinued the iodide of potassium for 3 days ; but had to recommence it without further delay ; for the symptoms of swelling and hardness of the tongue, running of saliva from the mouth, and adherence of the fibrous nodules to the tissues, began to rapidly increase, but abated with equal quickness under the renewed influence of the drug. The only untoward effect produced by these enormous doses was difficulty in staling, which appeared only after a month, and which soon passed off after the removal of its exciting cause. This colt took about 6 months to regain the use of his tongue sufficiently to enable him to eat oats efficiently. He regained perfect health, and was subsequently trained for racing.—TR.

under the heading "Pathogenesis" (page 205), it is in the highest degree improbable that actinomycosis is either contagious or infectious, in the sense that it can be transmitted from animal to animal, or from one of the lower animals to man. In fact, no indisputable case of infection of man by actinomycotic cattle has ever been observed. The official inspection of meat with regard to actinomycosis is therefore by no means so important as that applied to tuberculosis; especially as actinomycosis is usually only a local disease. The flesh of actinomycotic cattle may consequently be admitted for sale, as a rule, after the removal of the affected parts. Only in cases of general actinomycosis, which is very rare, should the sale of the meat be prohibited.

**Actinomycosis in Pigs, Horses, and Sheep.**—In *pigs*, actinomycosis appears in the lower jaw, larynx, lungs, wounds caused by the castration of males and females, mammae, flesh, and bones. When the bones are implicated, they become permeated with purulent cavities and sinuses, the contents of which show numerous yellow granules. Korsak frequently found in the tonsils of pigs the awns of barley which were covered with proliferations of actinomyces. The presence of general actinomycosis has been seen in pigs.

In *horses*, cases have been observed of actinomycosis of the bones, tongue, trachea, spermatic cord and submaxillary glands, the enlargement of which has been mistaken for that of glanders.

A few cases of actinomycosis of the lungs and muscles have been described in *sheep*.

**Actinomyces Musculorum Suis.**—Besides the ordinary fungus of cattle and swine (*actinomyces bovis*), Duncker has found in the muscles of the pig a second variety of the fungi which is club-shaped and which radiates from the centre. Duncker and Hertwig have called this fungus (which is met with only in the muscles) "*actinomyces musculorum suis*" to distinguish it from "*actinomyces bovis*." These authors do not claim that this ray fungus is identical with *actinomyces bovis*, with which it has apparently no connection. It has been found in a large number of pigs, and forms, when magnified 40 to 50 times, grey-brown foci of round, distinctly defined corpuscles. When magnified 300 times, we find in these foci micrococci-like formations, between which club-shaped filaments lie. The neighbouring muscular tissue is diseased, and the flesh is soft, very watery, and is on that account loathsome and unfit for human consumption. Hertwig advances the conjecture that the germs of the fungus are ingested during the summer months or at the commencement of autumn. Johnes distinguishes *actinomyces musculorum suis* from *actinomyces bovis* by the absence of the very characteristic blackberry-shaped tufts and by the fact that changes in the meat occur only in severe cases of the disease. According to Pfeiffer, we have here to deal with a myositis sarcosporidica, namely infection by Meischer's tubes (*sarcosporidia*).

**Botryomycosis in Horses.**—Rivolta was the first to find in the spermatic cord of castrated horses, fungi which he termed *discomyces equi*, and which he connected with *actinomyces*. These fungi were minutely described by Rabe as *micrococcus botryogenus*, and by Johnes as *micrococcus ascoformans*. They produce mycotic hypertrophy of the connective tissue, especially in the spermatic cord [scirrhus cord], skin of the neck and shoulders, and on the extremities (the so-called mycofibroma or mycoderma). They do not belong to the ray fungi, but to the micrococci. Bollinger, who found them also in multiple fibromatous nodules in the lungs of horses, proposed the name "*Botryomycosis*" ("grape-shaped" colonies) for this disease. Kitt obtained, from the cultivation from the botryomyces, tufts which closely resembled *staphylococcus aureus*, and found that their pure cultivations produced in the skin of a horse abscesses and proliferations which closely resembled microfibromata. Hell considers botryomyces to be simply tufts composed of pus cocci. He succeeded on every occasion in cultivating *staphylococcus pyogenes* from them. In horses and cattle, the botryomyces are found in the spermatic cord, skin, lungs, and not rarely in the udder. Observers have found among horses, new growths in the superior maxillary sinus and in the tail; multiple tumours extensively disseminated over the entire skin; and infection of the muscles, bones, pleuræ, connective tissue of the pelvic cavity, uterus, etc. This fungus is met with in the spermatic cord of pigs. It is stated that iodide of potassium exercises a specific curative effect on this disease (Thomassen, Sigmund, and others).

[See remarks (p. 213) on the employment of larger doses of iodide of potassium than are usually given.—Tr.]

**Actinomycosis of Man** chiefly occurs in the form of a suppurating inflammation and abscesses. The bones of the face and the tongue are the chief points of attack. From them, other organs such as the lungs, liver, kidneys, peritoneum, intestines and brain become affected by metastasis. As in cattle, the infection is probably transmitted exclusively by portions of plants which are studded with fungi.

#### STOMATITIS PUSTULOSA CONTAGIOSA OF HORSES.

**Nature.**—Stomatitis pustulosa contagiosa of the horse is an infective acute exanthema which chiefly attacks the mucous membrane of the mouth in the form of pustules and is distinguished by a benign course.

**History.**—From a careful study of veterinary literature, we find that stomatitis pustulosa contagiosa is by no means of recent date. Dard described, as early as 1840, a vesicular inflammation of the nasal mucous membrane which he had noticed in 1832 among 15 horses of the same battery, and

had at first considered to be acute glanders. The description of the disease, which lasts on an average 20 days and is non-malignant, leaves nothing to be desired. Bouley, Patty, Reynal, and Jakob reported exactly similar cases between 1840 and 1850. In 1856 Hering described in a voluminous treatise an epizootic outbreak of this exanthema, discussed its differential diagnosis with respect to acute glanders, and gave an account of inoculation experiments which he had made. About the same time König described the disease as *aphthæ ulcerosæ* of an apparently infectious character. In 1863, Vimercati reported on an equine mouth disease, by which 20 out of 90 cavalry remounts were attacked by a pustular dermatitis, stomatitis, rhinitis, and conjunctivitis. The publications of Palat and Silvestri on horse-pox undoubtedly referred to the same malady. Silvestri identified the complaint which he called "pox" as *impetigo labialis* of the German veterinary surgeons. The observations of Sondermann, Immelmann, Eggeling, and Haarstick refer indisputably to it. This disease gained renewed interest in 1878 by a publication of Eggeling and Ellenberger, who proposed to term it *stomatitis pustulosa contagiosa*.

**Etiology.**—*Stomatitis pustulosa contagiosa*, as the name implies, is an extremely infectious disease. The contagium is fixed and is contained chiefly in the saliva and mucus, and consequently the mucous membranes are far more susceptible to the action of the virus than the cutis. As a rule, it penetrates the tissues only at the site of a previous injury; though it may also be transmitted to horses by mere contact or by an intermediate bearer. In the order of comparative susceptibility, the horse comes first, then cattle, men, sheep, and pigs. Birds also contract the disease. Men occasionally become infected, especially attendants, veterinary surgeons, and children, on the hands, arms, conjunctiva, etc. The virulence of the contagium seems to weaken by inoculation from animal to animal. As a specific fungus has not yet been found to account for this disease; more exact researches are desirable. A previous attack appears to confer immunity on horses.

**Pathological Anatomy.**—The initial change consists in an inflammatory swelling of, and infiltration of small cells into the papillary layer of the skin, by which a microscopic formation of nodules takes place. In the further progress

of the disease, the nodules suppurate and liquefy in their centre ; the papillæ become destroyed ; and the entire thickness of the skin or mucous membrane, and even the subcutis or sub-mucosa may become involved. If, later on, a pustule which has sprung up in this way, opens, we shall find the tissue that surrounds the destroyed substance to be inflammatorily infiltrated. The healing of the ulcer takes place by the formation of granulations from below and by a new growth of epithelium from the edges. The cicatrix varies in size. About 12 days are required for complete recovery.

The foregoing changes are found chiefly on the mucous membrane of the oral cavity, namely, on the dental surface of the lips ; and on the frænum, tip, and sides of the tongue. They also attack the cutis and mucous membrane of the nasal cavities. The ulcers may be isolated, or may become confluent so as to cover a large area. If the process proceeds to the conjunctiva, a purulent conjunctival catarrh is usually the only unfavourable consequence. In rare cases, the formation of nodules on the mucous membranes is preceded by petechiæ. Vesicles, instead of nodules, may appear as forerunners of the pustules.

**Symptoms.**—With slight premonitory fever (temperature up to  $39.5^{\circ}$  C., and rate of pulse up to 60 a minute), the mucous membrane of the mouth becomes intensely red and hot, and isolated nodules are felt on passing the finger over its surface. Gradually, masses of mucus accumulate in the mouth, profuse salivation takes place, and the animal resists exploration of the mouth. As a rule, the general health is but little disturbed. The appetite is unaffected at first ; but later on, prehension of food becomes painful, and is effected with considerable salivation and even regurgitation. The temperature is seldom higher than from  $40^{\circ}$  to  $41.5^{\circ}$  C. After 2 or 3 days, the nodules increase in number and size, and their summits become pale and manifest the first signs of ulceration. In 4 or 5 days, the mucous membrane of the mouth becomes covered with ulcers in various stages of development. On the mucous membrane of the lips, tongue, and gums, we find conical prominences, which are 2 or 3 mm. high, and which are either isolated or in groups of from 2 to 7 or more in number. The apices of these prominences become always changed into circular ulcers. We can recognise in the ulcers a slightly depressed centre, which is excessively hyperæmic, shining, and

finely granulated. The ulcers are surrounded with a dirty-white or yellowish-white ring which looks like scalded epithelium, and which can usually be wiped off only with difficulty. By its removal, we expose the base of the ulcer, which readily bleeds, and which manifests the condition that was first observed in the centre of the undisturbed ulcer. We also find larger isolated ulcers and collections of confluent ulcers which frequently extend into the submucosa. Similar changes sometimes take place in the skin and especially on the upper lip, cheeks, margins of the *alæ nasi*, nasal mucous membrane, and even on more distant regions of the body, as for instance, on the skin of the fore-legs, and anus (infection transmitted by sponging), in which case it may happen that even the oral mucous membrane remains intact. Besides these local affections, there is usually great congestion of the mucous membrane of the nostrils, and of the conjunctiva, which may exhibit an intense purulent catarrh. There is at the same time, great swelling of the neighbouring lymph glands, particularly of the submaxillary glands and even of the glands of the shoulders.

The duration of the disease varies according to the intensity of the attack. The ulcers often begin to heal after a week has elapsed. The average duration of the entire course of the complaint is about a fortnight, with a maximum of about 3 weeks.

**Differential Diagnosis.**—It is not always easy to distinguish stomatitis pustulosa contagiosa from other somewhat similar processes; although in no case is the task impossible, if all the factors are taken into consideration. We should especially bear in mind the following diseases:—

1. *Acute Glanders and Farcy.*—Glanders (using the term as inclusive of farcy) has been frequently mistaken for the disease we are considering. Hering, 30 years ago, pointed out some differential diagnostic points between the two complaints. A mistake may be easily made, when, besides ulcers on the nasal mucous membrane and skin, and a nasal discharge, we find hard, painless swellings of the neighbouring glands; and when, by chance, no pustular changes of the oral mucous membrane are present, although the action of a contagium is evident. It is probable that formerly many horses affected with stomatitis pustulosa contagiosa fell victims to this diagnostic error. The following points distinguish stomatitis pustulosa contagiosa from glanders: the entire course of the

disease is benign; the symptoms of stomatitis are not those of glanders; the cutaneous ulcers of pustular stomatitis are not confined to the course of the lymphatics, as in farcy, the characteristic, rosary-like ulcers of which, with their indurated and eroded edges, are also absent; and the ulcers are circular, isolated, and frequently covered with a firm crust, under which they show healthy granulations and signs of a growth of epidermis commencing at their edge; these ulcers heal in about a week; the ulcers on the nasal mucous membrane are found only at the entrance of the nostrils and possess the same distinguishing differences, as regards glanders, as those of the skin; and inoculation produces stomatitis pustulosa, but not glanders. Also, the disease can be easily transmitted to cattle, especially to the mucous membrane of the vagina.

2. *Horse-pox*.—Next to glanders, horse-pox has probably been more frequently confused with stomatitis pustulosa than any other disease. Pustular stomatitis more closely resembles true equine variola, both clinically and anatomically, than any other disease, and especially any other exanthema. We must bear in mind, as an important diagnostic distinction, that equine variola is exceedingly rare, and that it occurs as a rule only on the pasterns.

3. *Follicular Ulceration*.—Formerly, under this term, stomatitis pustulosa was frequently mentioned. We may point out that the ulcers of stomatitis pustulosa are not connected with the anatomical position of the follicles, and may occur on places in which there are neither glands nor follicles.

4. *Stomatitis catarrhalis aphthosa, traumatica et ulcerosa* attack neither the skin nor the nasal mucous membrane; they manifest neither swelling of the lymph glands, nor the typical uniformity of stomatitis pustulosa; and they are not contagious.

5. *Vesicular eruption on the genitals and dourine* need scarcely be considered; for stomatitis pustulosa up to the present has produced its characteristic changes on the mucous membrane of the genital organs, only after artificial inoculation.

6. *Herpes Labialis* shows neither pustules nor ulcers.

7. *Canadian Horse-pox* is localised as a rule on the back (region of the saddle).

**Prognosis and Therapeutics.**—Prognosis is wholly favourable; because no recorded case has up to now ended fatally. For instance, about 1,000 horses became affected in the Prussian

army from 1887 to 1889 without any loss. As it, like all acute exanthemata, runs a typical course, no special treatment is necessary; although we may disinfect the ulcers with solutions of creolin, carbolic acid, alum, sulphate of iron, chlorate of potash, etc. As the disease is intensely contagious; it goes almost without saying that prophylactic measures should on no account be neglected.

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#### DIPHTHERITIC DISEASES.

General remarks—General remarks on diphtheritis of animals and diphtheria of man—Diphtheria in man—Croupy diphtheritic inflammation of the mucous membranes in fowl—Avian croupy diphtheritic inflammation of the mucous membrane probably caused by schizomycetes—Avian croupy diphtheritic inflammation of the mucous membrane caused by gregarines—Diphtheria of calves—Diphtheria of pigs.

**General Remarks.**—The name of diphtheritis is given to a particular form of inflammation of the mucous membrane which is characterised anatomically by a fibrinous exudation in the interior of the tissue with consequent necrosis, that is to say, a combination of exudation and mortification. By this definition we distinguish diphtheritic inflammation from croupy inflammation, which consists only of a superficial fibrinous inflammation of the mucous membranes with formation of croupy membranes that can be peeled off, without the deeper lying portions of the mucous membrane being implicated.

Diphtheritis of the mucous membranes is by no means a clinical unit; for it can be produced in animals by greatly-varying causes. Thus it may spring from purely mechanical means through pressure, as for instance, fæcal accumulation. Very many chemical substances, especially caustics, produce in the mucous membranes a form of a diphtheritic necrosis. A large series of infectious agents may also set up diphtheritic inflammation. We accordingly find diphtheritic processes during the course of rinderpest, malignant catarrhal fever in cattle, swine fever, acute glanders, petechial fever, "snuffing" disease of pigs (*Schnüffelkrankheit*\*), septicæmia puerperalis, gregarinosis of birds, and certain infectious diseases,

\* The word *Schnüffelkrankheit* appears to be used as a general term for difficulty of breathing due to stenosis of the nasal passages, whether caused by infectious rhinitis or rachitis.—TR.



such as diphtheritis of the mucous membrane of the nose and of the intestine, the causes of which, up to the present, have not been elucidated. Diphtheritic local affections may be produced experimentally in animals by the inoculation of different bacteria.

The same great variety in the clinical conception of this disease also exists with regard to human diphtheria. Therefore, in human medicine, a distinction is made between primary and secondary diphtheritis. The term primary diphtheritis or true diphtheria is applied to the specific, independent infectious disease, which is characterised by diphtheritic inflammation of the mucous membrane of the pharynx and which is produced by Löffler's diphtheria bacillus. Diphtheritic pharyngitis may occur secondarily to other infective diseases, such as scarlet fever, measles, smallpox, erysipelas, and whooping-cough, and is then called secondary diphtheritis or pseudo-diphtheria.

**General Remarks on Diphtheritis of Animals and Diphtheria of Man.**—The diphtheritic diseases of domestic animals are in no way related to human diphtheria. No indisputable case of the transmission of diphtheria to man from an animal has been proved. The cases recorded in medical literature, of the alleged transmission of infection, especially those of chicken-diphtheritis supposed to have been conveyed to man, are, on closer examination, reduced to mere assumptions, the forming of which has been due to entire ignorance of veterinary pathology. Even the statement of Gerhard—*which is the only one of the kind worthy of being quoted*—that in a chicken-hatching establishment, two-thirds of all the workmen attending on the fowl which were suffering from diphtheritis, became affected with pharyngeal diphtheritis, cannot be substantiated; for Gerhard did not observe the case personally. Considering the wide dissemination of chicken-diphtheritis, especially in hatching establishments, and presuming that this disease is identical with human diphtheria; a very great number of people would necessarily become daily infected, and reports on such cases would not be, as they are, extremely rare. We have examined thousands of domestic fowl and pigeons suffering from diphtheritis without having either seen or heard of a single case of such infection. On the other hand, no indisputable proof has yet been given that any disease similar to human diphtheria has been transmitted, either experimentally or accidentally, to any of the domestic animals.

**Diphtheria in Man.**—Contrary to the pathological nature of the diphtheritic ailments of domestic animals, which will be discussed later on under the headings of chicken-diphtheritis and calf-diphtheritis, human diphtheria is a specific acute infective disease which manifests itself locally in the form of a croupy diphtheritic inflammation of the mucous membranes, especially those of the pharynx and larynx. Löffler regards specific bacilli as the cause of human diphtheria. His diphtheria bacilli are stout rods about the length of a tubercle bacillus, double its thickness, and with rounded ends. They are found only in diphtheritic membranes. Their toxins become absorbed into the blood and produce, by means of it, serious general phenomena. Cultures of Löffler's bacilli are said to produce in rabbits and pigeons croupy membranes in the trachea, and sometimes grave constitutional disturbances and paralysis. Inoculated guinea-pigs die in a few days under symptoms of exudative pleuritis and œdema. The disease occurs chiefly among children up to their tenth year; it is very contagious; and begins probably as a local pharyngeal affection. After an incubatory period of from 2 to 5 days, symptoms of general disturbance (fever, malaise, and headache) first of all appear, and then difficulty in swallowing. The mucous membrane of the soft palate becomes greatly congested and the tonsils swollen. Dirty-white, firmly-attached covering membranes soon form on the mucous membrane of the pharynx (tonsils, uvula, and the palatine arch) with considerable swelling of the neighbouring lymph glands. The diphtheritic process then spreads to the mucous membrane of the larynx, where it causes stenosis of the larynx (dyspnœa and suffocation), and even to the mucous membrane of the bronchi and bronchial tubes. The symptoms of a septic general infection are also present. The chief complications are as follows: extension of the diphtheritic inflammation to the nasal cavity, oral cavity, tympanic cavity, conjunctiva, and intestinal mucous membrane; swelling of the articulations; myocarditis; nephritis; paralysis of the soft palate, vocal chords, muscles of the eye, muscles of deglutition, and, less frequently, muscles of the extremities; and ataxy. The mortality is very high. Fränkel, followed by Behring, was the first to begin the experiments which have been recently made for obtaining immunity against, and for finding a remedy for, diphtheria. Behring succeeded in curing, by the injection of the terchloride of iodine, guinea-pigs which had previously been given a lethal dose of diphtheria cultivations. The blood serum of these infected animals proved itself to be an absolutely certain agent for conferring immunity. Recently, Behring has begun to use horses for the manufacture of his diphtheria serum.

#### CROUPY DIPHTHERITIC INFLAMMATION OF THE MUCOUS MEMBRANES IN FOWL.

**General Remarks.**—The croupy diphtheritic inflammation of the mucous membranes in domestic fowl is not an etiological unit. On the contrary, we must distinguish at least two etiologicaly different varieties, which, although they possess nearly similar symptoms, are nevertheless different in their nature.

They may be described as follows: (1) a croupy diphtheritic inflammation of the mucous membranes, caused most probably by bacteria; (2) a croupy diphtheritic inflammation of the mucous membranes caused by gregarines.\*

Independently of microscopic examination, the croupy diphtheritic stomatitis which is produced by gregarines may be distinguished from that due to bacteria, by the fact that it can be transmitted by inoculation only with much difficulty. In spite of the great contagious influence of cohabitation, the disease has been directly transmitted by inoculation only in a very few cases. The inflammation of the mucous membranes caused by gregarines can, however, be transmitted by inoculation with the utmost facility.

#### AVIAN CROUPY DIPHTHERITIC INFLAMMATION OF THE MUCOUS MEMBRANES PROBABLY CAUSED BY SCHIZOMYCETES.

**Etiology.**—Speaking generally, this form is, next to typhoid, the most common and most dangerous epizootic among fowl. It has become generally known only during the last 30 years. Leisering, in 1860, appears to be the first veterinary author who described it. Zürn states that Russ in 1861 was the first to describe chicken diphtheritis. The existence of numerous publications by Italian and French veterinary surgeons referring to this disease, and other facts which we shall presently discuss, indicate that its starting point was in Italy and France, from which countries it was imported into Germany.

This affection was formerly considered by many to be of a tuberculous nature; but later on, it was regarded as true croup, true diphtheritis, or a croupy diphtheritic inflammation. As the fact had been proved that bacteria were frequently found in the inflammatory products, the view was generally adopted that these fission fungi possessed a pathogenic importance: a supposition which appears to be correct. Recently, various more or less contradictory theories have been advanced about other forms of fungi. Rivolta refers the cause of chicken-diphtheritis to two distinct species of fungi, which he calls “*epitheliomyces croupogenus*” and which develop only in the skin and mucous membranes, but not in the tissues or blood; thus entirely differing from the cocco-bacterial affection of the blood in diphtheria of man. In cases of croupy diphtheritic inflam-

\* Single-celled parasites which belong to the sporozoa.—Tr.

mation of the mucous membranes of pigeons, Löffler found in the different exudates and in the liver, numerous bacilli which were somewhat longer and thinner than those of the septicæmia of rabbits and which had rounded edges, besides several other kinds of fission fungi. Subcutaneous injections of pure culture of these rods produced necrotic inflammation, and in mice a characteristic infective disease. By the retro-inoculation of a pure cultivation which was obtained from the liver of a mouse, into two pigeons, diphtheritis of the oral cavity was produced in them. Löffler believes, however, that these bacilli should be demonstrated in a series of cases, before they can be looked upon as the true cause of pigeon-diphtheritis. Eberlein constantly found in diseased partridges, chain-like bacilli from 2 to 5  $\mu$  long and from 1 to 2  $\mu$  broad, which were rounded off at their corners and which contained spores.

**Occurrence.**—The inflammation of the mucous membranes caused by bacteria occurs, except in rare cases, only as an epizootic and attacks chiefly fowl and pigeons of the finer breeds, particularly those imported from Italy and France. German country fowl rarely suffer. Young birds are more liable to become infected than old ones. Parrots and water-fowl become attacked as well as gallinaceous birds (domestic fowl, turkeys, guinea-fowl, partridges, peacocks, and pheasants) and pigeons.

The chief cause of the large increase of the disease is the modern taste for fancy breeds, which has greatly stimulated the importation of foreign birds. Poultry shows, which have become more and more fashionable, certainly aid in the distribution of this malady; because sufficiently severe prophylactic regulations are not always applied to their management.

**Symptoms.**—Hardly any disease is so polymorphous as croupy diphtheritic inflammation of the mucous membranes caused by bacteria. Sometimes the mucous membrane of the mouth and pharynx is attacked; at other times, that of the nostrils and lining membranes of the accessory cavities. The affection may be limited to the mucous membranes of the eyelids and eyeball, or it may spread from the mouth into the larynx, bronchi, and alveoli of the lungs. In other cases, the alimentary canal may be the chief seat of the malady. The skin may also become involved. These respective local mani-

festations may appear singly or combined. The period of incubation lasts for a few days (p. 229).

1. *General symptoms.*—The general health, especially in mature, strong animals, is but little disturbed at the commencement of the disease. Differing from the course of human diphtheria, the local changes may be fairly well advanced before the illness of the patient attracts attention. We desire to lay particular stress on the fact, which has been denied by other writers, that the internal temperature, even when the symptoms are severe, never rises to an inordinate height, either at the beginning of the disease or during its progress. Anomalies in the distribution of the blood, defective nutrition and emaciation with their consequences, are to be observed during the further progress of the complaint. Thus we find that the collapsed erectile tissue on the head (comb and wattles) is dry and of varying temperature; the superficial heat of the body is unequally distributed; the visible mucous membranes are pale; and the feathers ruffled. The bird is off its feed; it gradually ceases to lay if it is a hen; it becomes depressed in spirits and feeble; droops its wings; makes only a feeble resistance when taken hold of; and likes to isolate itself. Before the fatal ending of the disease, the internal temperature sometimes falls, and the patient shows slight symptoms of brain irritation, quickly followed by cerebral depression.

2. *Croupy diphtheritic inflammation of the mucous membranes of the mouth and pharynx.* The first symptoms, which usually escape notice, are hyperæmia, and a slight swelling of the mucous membrane, which very quickly gives place to a deposit resembling hoar frost in appearance. This covering, which is thin at first, perceptibly increases in thickness, generally within 24 hours, and gradually changes into a white, fairly even, shining, caseous substance of a rather tough consistence, and very firmly adherent to its base. The thickness of this deposit, which soon assumes the aspect of a pseudo-membrane, may vary from 1 to  $1\frac{1}{2}$  mm. Its colour gradually changes to dirty yellow, and later on to brown. Its surface becomes rough and fissured; and its consistence, dry and brittle. The bird is usually obliged to breathe with an open beak, the head and neck being stretched out, and inspiration and expiration are very laborious. Prehension of food and deglutition are more or less seriously impaired. The favourite seats of the attack, which is not distributed over the entire surface of the mucous membrane, are the soft and hard palate with the cleft

of the palate; inferior surface of the tongue with the frænum and tip of the tongue; inner surface of the cheeks; angles of the mouth; and especially the neighbourhood of the superior portion of the larynx. From the angles of the mouth, the affection usually spreads to the skin. When the deposit has been removed—which, as we have already said, can be done only with difficulty—there always remains a more or less deep, uneven, greatly reddened or bleeding ulcer with eroded edges. We may notice at the base of this ulcer, various fine, villous proliferations, in which case isolated vascular loops project into the plates of the exudation, the removal of which gives rise to hæmorrhage. In the further progress of the disease, either the deposit becomes detached and a perfectly intact mucous membrane then becomes exposed to view, or ulcers resembling hard chancres, form, with probable loss of tissue and even necrosis, as for instance, that of the tip of the tongue.

3. *Croupy diphtheritic inflammation of the mucous membranes of the nasal cavity and neighbouring cavities.* At the beginning of the attack, we find in the neighbourhood of the nostrils, a serous fluid, which later on becomes dirty-yellow, greasy, and dries up, and then partly plugs up the nostrils and nasal ducts. If we press the nasal cartilages, we shall obtain the discharge of a fluid which is at first serous, and later on, more or less slimy, and is finally of a milky-purulent character. As a consequence of this discharge, the respiration becomes laborious and snuffling. The bird sneezes, by doing which, mucus is discharged, and at the same time, the patient frequently shakes its head. We find in the cleft of the palate inflammatory products, which we have already described. The process then very frequently spreads to the lining membrane of the cella infraocularis, which is more often unilaterally than bilaterally affected. Under the median angle of the eye and behind the root of the beak, there is a protuberance of the soft parts, which is hyperæmic and collaterally œdematous, painful to the touch, and generally hot. Pressure on it produces an abundant discharge from the nostril of that side. This swelling goes on increasing, spreads chiefly under the eyeball and towards the rear, and may finally attain the size of half a walnut. At the same time, the corresponding half of the hard palate increases to at least double its width, and bulges out into the oral cavity. In its further progress, the tumour over the cella infraocularis becomes tense and firm. If opened early it will discharge a thick, creamy fluid, or a soft, cheesy substance. Later on, the dirty-white or yellowish contents become

dry and crumbling, or present the appearance of tough, thin membranes or flakes lying closely together. By enormous accumulations of the dried-up exudate, the before-mentioned sinus frequently becomes dilated to such an extent that it may attain a diameter of 2 or 3, or even 4 cm. The continually-increasing pressure causes displacement of the neighbouring soft parts, especially of the eyeball, other half of the palate, etc. ; and osseous atrophy with absorption, in consequence of which the head becomes greatly disfigured and misshapen. The bird is often hardly able to open its beak, or to feed.

4. *Croupy diphtheritic inflammation of the mucous membranes of the larynx and trachea.* Considerable exudative accumulations take place in the neighbourhood of the opening of the larynx on the mucous membrane which lines the larynx, and on the upper part of the trachea, which can easily be examined in birds, especially in large domestic fowl ; the resulting dyspnœa being proportionate to the degree of stenosis. The breathing is deep and laborious ; the air being, as it were, pumped up ; and the beak is held wide open. The bird gives utterance to peculiar whistling, singing, or wheezing noises, combined with moist râles, coughing, and panting, and frequently dies of suffocation. The coughed-up matters partly accumulate in the pharynx and partly adhere to and soil the throat and breast. Their decomposition gives rise to the disagreeable, sickly smell which we may perceive, even when at a considerable distance from the affected birds.

5. *Croupy diphtheritic inflammation of the eyes.* Catarrh of the eyelids is the first symptom of this complaint. The initial hyperæmia of the conjunctiva soon changes into a muco-purulent catarrh, the discharge of which collects at the median angle of the eye and flows away. The eyelids and their neighbouring parts become œdematous and hot ; and the eyelids have a great tendency to stick together. By forcibly pulling them open, vent is given to masses of inflammatory products, which are at first of a serous and muco-purulent nature, but soon become caseous and then assume (corresponding to the form of the sac made by the eyelid and eyeball) a semi-lunar or lenticular shape. If the eye is left to itself, either the eyeball becomes atrophied by the pressure of the accumulated discharge, or, as very often happens, the diphtheritic process spreads to the internal parts of the eye and causes in it severe destructive changes (panophthalmia). At first, the disease spreads from the conjunctiva scleræ, to the cornea,

which becomes affected by a superficial, smoky opacity, and which subsequently becomes swollen and opaque from parenchymatous inflammation (keratitis parenchymatosa), in which case a firm cone of exudation of varying size often quickly forms on the thickened, and now perfectly opaque cornea, and presses the eyelids asunder. Finally, suppurative perforation of the cornea and purulent panophthalmia with subsequent destruction of the eyeball, occasionally take place.

6. *Intestinal affection due to croupy diphtheritic inflammation of the mucous membranes.* This affection of the intestinal canal consists in a more or less extensive catarrhal inflammation and croupy diphtheritic infiltration, which, as a rule, occurs only when one of the previously described local maladies has been present for a considerable time, and usually ends the course of the disease. Zürn states that it occurs early, as an independent complaint, in water-fowl and turkeys. In addition to an increase in all the general symptoms, diarrhœa sets in; the fæces being foetid, very thin, pappy or mucilaginous, and sometimes mixed with blood or bloody pus. Zürn states that this diarrhœa causes excessive debility, dulness, and stupor, and, when it appears in a severe form, is a forerunner of death.

7. *Affection of the skin in croupy diphtheritic inflammation of the mucous membranes.* The transmission of the croupy diphtheritic affection to the skin appears on the eyelids and corners of the mouth only to a small extent in the form of cutaneous ulcerations. Similar changes may also be found in the neighbourhood of the anus and on the lining membrane of the auditory meatus.

**Pathological Anatomy.**—The *post mortem* examination of the usually emaciated and anæmic cadaver sometimes shows, besides the already described changes which could be demonstrated during life, accumulations of whitish-yellow secretion in the bronchi, which appear to be filled with firm, crumbling masses, whilst the parenchyma of the lungs manifest signs of atelectasis or œdema. More frequently we find simple tracheal and bronchial catarrh with more or less extensive catarrhal inflammation of the lungs. Cellular fibrinous pericarditis, subepicardial ecchymoses and cloudy swelling of different organs, such as the liver, are by no means rare. The catarrhal inflammation of the intestinal canal is often confined to the upper and anterior parts. Zürn sometimes found the cæcum and rectum completely filled with yellow, stratified



masses which adhered firmly to the intestinal mucous membrane ; and that the intestinal mucous membrane was frequently ulcerated. All authorities are agreed on the results of the microscopical examination of the excreted matters. The soft caseous discharge from the conjunctiva consists, for the greater part, only of round cells which cannot be distinguished in any way from degenerated leucocytes. They vary in length from 0.006 to 0.009 mm. and are distinguished by their great tendency to become rapidly broken up. Usually, a large number of bacteria, chiefly micrococci, are also present. When the inflammatory products are treated with a watery solution of methylene violet, the uniformly small micrococci may be seen in enormous numbers. The same result is obtained by the examination of the more recent aggregations in the mouth and pharynx, except that pavement epithelium in the exudate is more abundant in them and in the cavities of the head, bronchi, etc. The before-mentioned round cells quickly change, even in quite freshly excreted masses, into a fine granular detritus, so that the results vary according to the age of the discharge.

**Course.**—The course of this croupy diphtheritic inflammation of the mucous membranes is always slow, and generally chronic. It usually lasts for weeks and even months, until the affected birds succumb to the disease. Only young birds, especially pigeons, form an exception to this rule ; for they die of the disease in a short time. These facts agree with the few inoculation experiments which, up to the present, have been successful. Trinchera observed after inoculation with nasal mucus that the period of incubation was from 7 to 20 days, and that the symptoms slowly increased in intensity for from 8 to 15 days. He found that strong animals recovered their health in from 60 to 70 days. Krajewski states that the period of incubation in domestic fowl is from 4 to 10 days. According to the experiments of Babes and Puscarin, it lasts only 2 or 3 days in pigeons.

**Prognosis** is generally unfavourable. The comparatively hopeful cases are those of pigeons ; of acclimatised, strong, well-nourished fowl ; and of birds in which the process is limited to the pharynx. As a rule the disease is difficult to successfully combat, and supposed recoveries are often only apparent. Thus it happens that birds which are regarded as cured may, on being returned to their usual haunts, prove a cause of infection to their

healthy companions ; and the disease consequently acquires an insidious character. The average mortality is from 50 to 70 per cent.

**Therapeutics.**—*Prophylaxis* plays an important part in the croupy diphtheritic inflammation of the mucous membranes. It consists in exact examination ; maintenance of quarantine for several days' duration with every newly-bought bird ; abstaining from exhibiting birds at those poultry shows which are not under veterinary superintendence ; carefully observing each bird ; paying attention to suspicious symptoms, especially nasal discharge, tears, etc. ; segregation of the affected from the healthy ; scrupulous cleanliness in coops and cages ; periodical disinfection with carbolic acid, corrosive sublimate, creolin, lysol, etc. ; and destruction of the bodies of birds which have died from the disease, and of the utensils that have come in contact with them.

If in spite of the foregoing precautions, the disease has once broken out, its course may be favourably influenced by early treatment. Careful nursing is more efficacious than medicinal agents, among which we may mention the following disinfectants : carbolic acid, creosote, tar, creolin, lysol, chlorate of potash, permanganate of potash, corrosive sublimate, chloride of lime, tincture of iodine, salicylic acid, boric acid, sulphate of iron, liquor ferri perchloridi, sulphate of zinc, tannin, oil of turpentine, caustic potash, nitrate of silver, lactic acid, etc. These have been used either singly or in various combinations.

We have found the application by a brush, of a solution of corrosive sublimate (1 or 2 in 1,000), or one of creolin (1 or 2 in 100), to be very effective when the disease is localised. Other practitioners have also frequently obtained good results with creolin. We can recommend the removal of the pseudo-membranous inflammatory products, especially in the case of pigeons, only when it can be done easily, and without causing hæmorrhage. Our own experience prompts us to deprecate forcible removal and cauterisation of the base of the ulcer. The tumours over the cavities of the head should be incised, their contents removed, and the frequently severe hæmorrhage prevented by inserting a cotton-wool tampion saturated with liquor ferri perchlor. We have found fumigations with tar especially effective in relieving the affection of the air passages. Zürn recommends internally and externally a mixture of 150 grammes of a decoction of walnut leaves (15 grammes to a litre of water), with 20 grammes of

glycerine, 5 grammes of chlorate of potash, and  $\frac{1}{2}$  gramme of salicylic acid in 15 grammes of rectified spirit. Of this we may give once or twice daily, from a teaspoon to a tablespoonful to large birds ; and from a quarter to half a teaspoonful to pigeons. We should also paint the parts with this solution twice or thrice daily. For the intestinal complaint we recommend tannin or sulphate of iron (in a 1 to 2 per cent. solution or in pills made up with butter and white bread ; for pigeons 0.2 to 0.5 gramme ; fowls 0.5 to 1 gramme ; and geese 0.5 to 2 grammes *pro dosi*).

#### AVIAN CROUPY DIPHTHERITIC INFLAMMATION OF THE MUCOUS MEMBRANES CAUSED BY GREGARINES.

**Etiology.**—Gregarines or psorospermia, which are called coccidia when they are enclosed in a capsule, are exceedingly minute protozoa, consisting of small lumps of protoplasm, and possess the power of amœboid movement in their early stage. In their further development they become encapsuled. In shape they may be cylindrical, spherical, oval, or elliptical. They are very widely disseminated as parasites in the animal kingdom ; and are found not only on birds, but also in rabbits, rats, dogs, and even on fish, snails, and earthworms. The history of the development of these interesting parasites, which always live together in countless numbers (hence the name gregarines, *i.e.*, animals that live in herds), has been elucidated chiefly by Eimer and Leuckart. In their early stage they are naked inhabitants of epithelial cells. By penetrating into the cells of the mucous membrane of birds and by filling up these cells (in many cases completely, so that the enclosed gregarines assume the appearance of large nuclei), they produce grave disturbance of nutrition which tends to induce necrosis of the parts attacked. Rivolta and Silvestri were the first who traced to psorospermia (naked gregarines) an epizootic which broke out amongst fowls in the neighbourhood of Pisa in 1872, and which these authorities personally observed.

Gregarinous croupy diphtheritis is distinguished by the ease with which the disease spreads from the mucous membrane of the mouth to the skin of the head. It can be readily transmitted artificially, and in general is not difficult to cure, especially when it is confined to the mucous membrane of the mouth, pharynx, upper part of the larynx, and skin.

**Symptoms.**—The clinical phenomena of avian gregarinous

diphtheritis agrees in all particulars with those of bacterial diphtheritis when these respective complaints affect the mucous membrane of the head. Here, also, the symptoms are essentially those of croupy diphtheritis of the mucous membrane of the mouth, pharynx, air passages of the head, larynx, conjunctiva, etc., with secondary intestinal affections. Gregarines may also give rise to primary and independent enteritis (Zürn).

In gregarinous diphtheritis, the *skin* is much more frequently implicated than in diphtheritis caused by bacilli. The cutaneous affection consists of hypertrophied nodules on the skin, which are known as gregarinous epitheliomata (*epithelioma gregarinosum* of Bollinger, and are identical with the *mol-luscum contagiosum* of man). Their favourite seats are those parts of the head that are not covered with feathers: root of the beak, neighbourhood of the nostrils, angles of the mouth, lobes of the ear, parts adjacent to the external auditory meatus, wattles, surface of face, edges of the eyelids, intermaxillary space, and especially the comb. They sometimes spread over the feathered parts of the head, throat and neck, and may occur on the outer surface of the thighs, abdomen, under the wings, and in the vicinity of the cloaca. At first, these epitheliomata appear in the skin, as flat nodules, which soon become prominent, and which vary in size from a poppy seed to a millet seed. Later on, they usually attain the size of a hemp seed. They are of a reddish-grey or yellowish-grey colour; often show distinctly in their earlier stages of development a peculiar greasy nacreous lustre; and are rather firm to the touch. Their surface soon becomes covered with a dirty-grey, yellow-brown, or red-brown crust. They are discrete and disseminated in considerable numbers on the erectile tissues, etc. They vary in size according to their age; and frequently lie rather close to one another, so that the affected parts look as if coarsely granulated; or they are crowded together in such a manner as to give the appearance of large warts with divisions through them, or of mulberry-like hypertrophies. Even single nodules, to say nothing of groups, may attain the size of a lentil, pea, cherry-stone, broad-bean, or larger object. The older they become, the rougher, and more covered with knobs will be their incrustated surface.

If the edges of the lids of the *eyes* be affected by these tumours, they will become nodular, swollen and closed. The conjunctiva in this case also suffers; it projects outwards; becomes catarrhally inflamed; assumes a yellowish colour at

the seat of eruption; and its surface becomes covered with crusts. Purulent conjunctivitis may appear, and the inflammation may spread to the sclerotic and cornea, with keratitis and panophthalmia as the result. If, as sometimes happens with pigeons, the eruption of nodules extends over the whole of the skin of the eyelids and its neighbourhood, the entire eye will become covered with mulberry-like proliferations of various sizes.

**Course and Prognosis.**—The course of this gregarious disease is, on the whole, more benign than that of diphtheritis caused by bacteria. The epithelioma of the skin often heals spontaneously, and the nodules dry up and fall off. Gregarious inflammations which are restricted to the mouth, pharynx, and entrance to the larynx, also heal spontaneously at times, or under simple treatment. Other cases, however, run a fatal course, either directly by suffocation (localisation of the process in the larynx and trachea), or from cachexia brought on by extension of the eruption and by increase of the catarrhal inflammation, especially when it involves the intestines. Loss of appetite, depression of spirits, emaciation, ruffled condition of the feathers, etc., are also observed in such instances. Bollinger noticed that fowl died 4 or 5 weeks after the commencement of the infection, and 3 or 4 weeks after the first nodular eruption had appeared.

**Pathological Anatomy.**—If we examine the products and detritus of the croupy diphtheritic inflammation, we shall find in them, cast-off epithelial cells which contain in their interior a rounded body that fills up half or more of the cell-space, and looks like a greatly enlarged nucleus. These bodies are highly refractive, and have a greasy, glassy lustre, and swollen homogeneous appearance. These spherical formations are also found free and in varying numbers in the croupy diphtheritic excretions.

If we pass on to the examination of the tissue of the affected mucous membranes, we shall also find (in teased-out preparations) the individual epithelial cells to be in the same condition as that just described, as far as the opaque, stratified, cellular excretion and the coagulative necrosis of the cells will allow us to observe. They all contain the same strikingly large, rounded bodies.

The microscopical examination of the epitheliomata (for-

merly wrongly called "bird-pocks") shows in fresh sections and teased-out preparations, large masses of heaped-up and considerably enlarged epidermic cells containing exactly the same round corpuscles as the proliferated epithelium of the affected mucous membrane of the mouth. If we harden in alcohol one of the smaller and more recent nodules of the epithelioma, and if we make sections at right angles to the surface, we shall find on examination that the nodule consists of enormously hypertrophied and considerably enlarged epithelial cells. All the epidermic cells, with the exception of the most recent, contain the rounded bodies which resemble greatly swollen\* nuclei and which are oval in the uppermost layers, like the cells themselves. Their peculiar fatty lustre and swollen aspect increases according to the age of the cells. Although in the youngest cells, these corpuscles respectively occupy scarcely a quarter of the interior, they soon grow to double that size or even more, and fill up the entire cavities of the cells in the outer epithelial layers, in which, on account of their highly glassy lustre, each invaded cell looks like a single large vacuole. The connective tissue which lies under the stratum-mucosum is generally hyperæmic and infiltrated with some small cells. We can also demonstrate in some parts of the tissue of the epithelioma a small cellular hypertrophy as an accumulation of nuclei. Respecting these last-mentioned conditions, particularly as regards the contents of the cells, we obtain exact information only by the use of certain stains, of which picro-carmin is the most useful. It colours the cell-nuclei a bright brown-red, and the rounded bodies (the naked gregarines), yellow. If we examine the *stratum mucosum* by means of a stained section, which should be as thin as possible—not more than one cell-layer thick—we shall find that all the youngest spindle-shaped epidermic cells contain elongated oval nuclei with one or more nucleoli. If we proceed from here to the neighbouring cell-layers, we shall quickly perceive very young epidermic cells, which, besides their still perfectly well-preserved, brown-red stained, and normally large nucleus, already contain a yellow-stained, rounded, homogeneous, small lump of protoplasm which is not quite as large as a leucocyte. Such invaded cells are also distinguished by being of a much greater size than the intact neighbouring cells and by appearing as if they were inflated.

\* The word translated here by "swollen," means burst from excessive swelling, like what we may see in a well-boiled, "mealy" potato.—TR.

**Therapeutics.**—The treatment of gregarinous diphtheritis is the same as that of diphtheritis caused by bacilli. We have obtained particularly good results by painting the affected mucous membrane with a 1 to 2 per cent. solution of creolin or carbolic acid in equal parts of glycerine and water (*creolin* 5, *glycerini*, *aquæ destillatæ* āā 100). Pure glycerine alone kills the gregarines by depriving them of water. Glycerine may be also given internally, especially to geese, in doses of a teaspoonful up to a tablespoonful in cases of gregarinous enteritis (Zürn).

A third variety of croupy diphtheritic inflammation of the mucous membranes is caused by *cercomonas* [microscopic parasites (infusoria) of about 0.01 mm. in length. They are pointed at one end, and have a long flagellum at the other end.] Rivolta found in the deeper layers of the white-yellowish plaques of fowls that were suffering from croup of the pharynx, œsophagus, and crop, thousands of oval cell-shaped infusoria which possessed the power of exceedingly rapid movement. He gave them the name of *cercomonas gallinarum*, and considered that they were the cause of pseudo-croupy inflammation. Zürn observed *cercomonas* in pigeons, in which he found that the inflammation caused by them was only slight, and that the yellowish, gelatinous, easily-liquefying coatings of the mucous membranes were only slightly adherent. We cannot agree with Zürn's view that micrococci and bacteria may form out of the gregarines of the gregarinous variety by disintegration, and that these micrococci and bacteria produce the former variety, so that consequently both varieties are only different stages of development of one and the same disease; for we have never been able to find gregarines in diphtheritis due to bacteria, which task would have been very easy had they been present. Equally little are we able to accept Pfeiffer's theory that the diphtheritis of fowls is a uniform disease caused by flagella (protozoa). Babes has shown that the flagella are parasites of the normal mucous membranes of birds, and are by no means pathogenic. The parasitic nature of the gregarines in diphtheria of birds is denied by others, who state that the supposed gregarines are only changes of the nucleus in consequence of cell degeneration (Virchow, Kromayer, and others).

#### DIPHTHERIA OF CALVES.

**Etiology.**—Dammann and, more recently, several others have described, under the name of "diphtheria of calves," a diphtheritic inflammation of the mucous membrane of the mouth and pharynx, which seems to occur not only among calves, but also among lambs. The true cause of it is unknown. This disease has not been very accurately differentiated from other diseases owing to the fact that it closely resembles the diphtheritic form of stomatitis and pharyngitis, and also foot and mouth disease. Consequently it is not improbable that two or more

diseases may be included under the name of diphtheria of calves. Dammann considers that the diphtheria of calves is identical with the diphtheria of man, and that both diseases are produced by one and the same micrococcus. He supposes that calves and their sheds are the hitherto unknown source from which men obtain the diphtheritic poison; a conjecture which has no scientific foundation. Vollers regards the bacteria which exist in the air immediately above the floor of the cowshed as the cause of the disease, and maintains that the decomposition of the urine in the litter and the transformation of the urea into carbonate of ammonia, which is irritating to the mucous membrane, are the chief predisposing causes. Dammann believes that this complaint is contagious, and that it can be transferred by inoculation to lambs, rabbits, and man. It attacks animals particularly during their first few weeks of existence, but never when they are grown up. It is said that the period of incubation is very short, but that the infectious matter remains virulent for a very long time.

Löffler, who investigated diphtheria of calves for the German Imperial Board of Health, believes that the specific cause of the disease is bacilli, and not micrococci, as Dammann assumes. He found on the inner border of the tissue destroyed by this disease, large, long bacilli which formed undulating threads, and which differed entirely from the *bacillus diphtheriæ* of man, both by their form and by their action on the infected parts. Ritter confirmed these statements. Kitt regards the cause of the diphtheria of calves to be the bacillus of necrosis, which he considers identical with Löffler's bacillus.

**Pathological Anatomy.**—The principal changes in diphtheria of calves are found in the mucous membrane of the mouth and pharynx. This affection shows itself chiefly on the tongue, hard palate, and inside of the cheeks, as a yellow, croup-like deposit which is partly circumscribed, partly diffuse, and which adheres closely to the underlying tissues. It sometimes attains a thickness of a few centimetres. On many places, the croupy membrane entirely destroys its underlying tissues, such as the lamellæ of the bones of the hard palate, and the muscular apparatus of the tongue. These deposits, when examined microscopically, are found to be composed of dense accumulations of micrococci, detritus, threads of fibrine, leucocytes and the long bacilli of Löffler. Similar changes, though to a lesser degree, are found in the nasal cavity, larynx, trachea, and intestinal



canal. Koudelka found in the larynx a greasy, drab-coloured (grey-yellow), burnt-smelling coating, on the removal of which a deep ulcer extending to the cartilage and having an uneven base was disclosed to view. In one case, the interdigital skin of both fore-feet showed an enormous deeply-penetrating diphtheritic infiltration. On *post mortem* examination, besides the above-mentioned changes, circumscribed, pneumonic foci were found in the lungs with suppuration and purulent pleuritis. The spleen was not enlarged.

**Symptoms.**—The disease during life closely resembles foot and mouth disease. There is entire loss of appetite, profuse salivation, purulent nasal discharge, and fever. The cheeks and laryngeal region become swollen; the patient coughs; and the previously described changes are to be found in the oral cavity. Difficulty of breathing sets in later on, and sometimes diarrhœa. There is great debility, marked inclination to continue recumbent, and rapid emaciation. Death, which is the almost invariable termination, generally takes place in 4 or 5 days; although the period may be prolonged to 2 or 3 weeks in lung or intestinal cases.

**Differential Diagnosis.**—A distinct differentiation of the diphtheria of calves from foot and mouth disease is difficult. Dammann states that only calves, as we have already said, are attacked by this epizootic during the first weeks of life and that older animals are immune. Besides, the course of the diphtheria of calves is stated to be more acute and more malignant than that of foot and mouth disease.

**Therapeutics.**—*Prophylaxis* consists first and foremost in separating the infected animals from the healthy and in a thorough disinfection of the shed. The croupy deposits in the oral cavity should be treated with disinfecting agents, such as carbolic acid, creolin, lysol, salicylic acid, and chlorate of potash, as already stated. If possible the deposits should be removed, and the mouth repeatedly washed out. Dammann recommends the frequent injection into the oral cavity of a half per cent. solution of carbolic acid, or the application, by means of a brush, of a paste made of salicylic acid and water, and the internal administration of salicylic acid.

**Diphtheria in Pigs.**—Johne observed diphtheria of the upper portions, respectively, of the digestive canal and air passages (glossitis,

tonsillitis pharyngitis, and laryngitis follicularis diphtheritica) in several pigs which had been sent together by rail and which had shown great difficulty of breathing and prostration. The most striking changes were found in the tonsils, which were considerably swollen on both sides. The openings of the amygdaloid cavities were pretty well filled with yellowish crumbling plugs, which could be squeezed out. The section surface appeared yellow, and resembled dry cheese. The neighbouring mucous membrane was stuffed with numerous rounded, grey-yellow [drab-coloured] deposits, which were the necrotic solitary follicular glands, and which were as big as a pea. The bacillus of necrosis was found in the necrotic mucous membrane.

According to Kitt, there occurs sporadically and enzootically an independent diphtheria of pigs which has no connection with swine fever. He states that it is caused by the bacillus of necrosis, which occurs also in diphtheria of calves. The mucous membrane of the tongue, cheeks, pharynx, and stomach shows yellow-white caseous deposits; and that of the small intestine and colon, diphtheritic necrosis.

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#### PARASITIC STOMATITIS.

**Etiology.**—This disease, which can be transmitted by man to animals, is a mycosis of the mucous membrane of the mouth and pharynx, and sometimes also of the entrance of the larynx and the upper part of the œsophagus. It is caused by a fungus, which was discovered in 1840 by Berg and Gruby, and which was formerly known as *oidium albicans*. Gravitz considered this fungus to be identical with *mycoderma vini*. More recent investigations by Plaut have shown that it is a very common mould fungus, *monilia candida*, which grows on fresh cow-dung, rotten wood, and thrives in uncooked cows' milk and on nutritive mediums containing sugar. Plaut has cultivated the fungus and produced the disease in fowls by inoculating pure cultivations. The fungus is found in human beings (sucking babies), calves, foals, and especially in birds. It forms a mycelium, which consists of filaments and roundish, oval or cylindrical shining cells (*gonidia*), and can be stained with Gram's solution.

**Pathogenesis.**—The settlement of the fungus on the mucous membrane of the oral cavity depends on certain circumstances which have not yet been fully ascertained. Youthfulness, constitutional weakness, gastric disturbances, defective cleaning of the mouth, and feeding on milk and on food containing a large proportion of starch, seem to favour the growth of the fungus.

Decreased power in chewing and in swallowing appears to assist the adherence of the fungus to the mucous membrane of the mouth, by the prolonged retention in the mouth of food substances which are inclined to form acids. In human beings, suspicion is especially directed to the mother's milk, a portion of which may be left in the oral cavity of the baby, or may be brought into prolonged contact with the mucous membrane of the mouth by frequent vomiting. Martin observed a case of the fungus being transmitted from a child to a fowl.

**Anatomy.**—The local affection appears to be restricted in fowls to the mucous membrane of the œsophagus and crop. We notice first of all, on the otherwise quite intact mucous membrane, small white spots, which become enlarged and confluent. They finally form a white, grey, or yellow substance, which is frequently divided by clefts into compartments, and which is of a crumbling or greasy, croup-like consistence. If we remove this membranous deposit, we shall find that the underlying mucous membrane is reddened, or that it has undergone slightly ulcerous changes.

The microscope shows that the described deposits on the mucous membrane are composed of cast-off pavement epithelium, between which the fungus penetrates in the form of fine, decussating filaments, that are plaited into a dense felt. On the ends of these filaments we can recognise the formation of spores, as well as free, spherical or oval, green spores.

**Symptoms.**—The symptoms in fowl, with the exception of the before-mentioned anatomical changes, are not very characteristic. The affected birds are depressed in spirits, and become emaciated in spite of their possessing a voracious appetite ; the crop is distended ; a sour smell comes from the mouth ; and death ensues with violent convulsions.

**Differential Diagnosis.**—This stomatitis may very easily be mistaken, as often happens, for other inflammations of the oral mucous membrane ; for croupy diphtheritic stomatitis, especially in fowls ; and for aphthous, pustular, and ulcerous stomatitis in mammals. The essential data for correct differentiation can be furnished only by the microscope.

**Therapeutics.**—Besides strengthening the patient with good food and by overcoming contingent gastric disturbances,

we should be most careful to cleanse the oral cavity and to keep it aseptic. Plaut recommends, in particular, the painting on of a solution of corrosive sublimate (1 to 1,000), as well as the removal of the deposit.

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### TETANUS.

**Nature.**—Tetanus is an infective disease caused by a specific bacillus. Kitasato, in 1889, isolated the tetanus bacillus which was described in 1884 by Nicolaïer, and obtained a pure cultivation. The tetanus of man, the tetanus of animals, and the tetanus which can be produced experimentally by inoculation with soil, are respectively caused by one and the same pathogenic organism, that is, by the bacillus of Nicolaïer.

**Bacteriology.**—The tetanus bacillus, in its spore-containing condition, forms, according to Kitasato, rods in the shape of a pin or ladle with a terminal spore 6 to 8  $\mu$  long. In its sporeless form, it consists of rods which are just the same length, are straight and rounded off at the ends, and appear singly or in groups from two to four in number. These rods possess a weak automatic power of movement and form long, slightly undulating pseudo-threads. The tetanus bacilli stain well with the customary aniline colours, and also by Gram's method. For the spore containing bacilli, Ziehl's double staining may be used. They are obligatory anærobes and consequently grow only when air is excluded, and better still in hydrogen. Cultivations thrive in slightly alkaline, pepton-containing gelatine, agar-agar, and broth. An addition of grape sugar (2 per cent.) or sulph-indigotate of soda (1 *per mille*) accelerates their growth. The cultivations are characterised by their empyreumatic smell. The single colonies have a certain similarity with cultivations of hay bacillus. A dense centre is surrounded by a fine, uniform wreath of rays. The gelatine slowly liquefies with formation of gas. Tetanus bacilli thrive best in a temperature of from 36° to 38° C. Their growth stops at a temperature lower than 14° C. At the temperature of the blood, spores form in 30 hours; and with one of from 20° to 25° C., in not less than a week.

*The resistance of tetanus bacilli*, especially in the spore form, is very great. Thus, for instance, the action of a 5 per cent.

solution of carbolic acid continued for 10 hours, has no effect on the spores (Kitasato). Dried spore-containing tetanus-pus retains its virulence for over 16 months (Kitt). Clams used for castration may remain capable of transmitting the virus of tetanus to horses for eighteen months, even after they have been placed for 5 minutes in boiling water, or in a 4 per cent. carbolic solution (Nocard). On the other hand, the spores are killed by being exposed to a temperature of 100° C. for from 2 to 5 minutes in a steam sterilizing apparatus. According to Tizzoni and Cattani, mineral and organic acids, even when concentrated to the highest degree, produce no effect whatever on the spores. The spores in the cultivations are, however, killed by a 1 per cent. solution of nitrate of silver in one minute; by a one-tenth per cent. of the same agent in 5 minutes; by a 1 per cent. solution of corrosive sublimate in 25 minutes; by a 5 per cent. solution of creolin in 5 minutes; by a 1 per cent. solution of permanganate of potash in 10 minutes; by a 5 per cent. solution of carbolic acid mixed with  $\frac{1}{2}$  per cent. of hydrochloric acid, in 25 minutes; and by a solution of iodine in 6 minutes. Chlorine, chlorinated lime and tar possesses a destructive action on the spores, which, however, resist putrefaction for a long time. Iodine trichloride seems to possess a particularly strong antiseptic effect on the tetanus spores (Behring). The spores are exceedingly resistant to the digestive fluids, especially to the gastric juice. Sormani states that they may grow and increase even in the intestine.

[Carougeau (Cadéac's *Encyclopédie Vétérinaire*) tells us that in a state of nature, the microbes of tetanus exist in the form of spores which do not manufacture any poison. But if they are introduced into the animal tissues under certain favourable conditions, they go through a form of development and subsequently secrete their toxins, with the result of setting up this disease. If these spores are inoculated by themselves into healthy tissue, they are unable to produce their toxins for the time being, and will in all probability be destroyed by the leucocytes. Some of them may, however, remain alive though inactive for even 3 months or more, and, under conditions favourable to their development, they may subsequently become virulent and produce tetanus. Hence the fact that occasionally the period of incubation is unusually long. The conditions in question appear to be those which diminish the resistance of the tissues, by repelling the protective leucocytes; one of the chief of these conditions being the presence of certain common microbes which produce pus, and consequently favour the development of the micro-organisms of tetanus. Suppurating wounds are therefore particularly good breeding grounds for the bacilli of this disease. Injury and the presence of foreign bodies also act favourably in the same

direction. Hence, we find that wounds which become polluted by soil and dirt are specially liable to be followed by symptoms of this disease ; and the lesson is obvious that the cleaner we keep wounds, the less danger will there be of tetanus.—TR.]

**Pathogenesis.**—Like the bacillus of malignant œdema, the bacillus of tetanus is an ubiquitous bacterium. Numerous inoculation experiments have proved that it occurs everywhere in garden mould and is not restricted to any geographical limit. The inoculation of garden mould from Berlin, Göttingen, Wiesbaden, and other places, in mice, guinea-pigs, and rabbits, was found to produce tetanus of a uniform type. Mould rich in animal manure, especially horse-dung, forms a particularly favourable abode for tetanus bacilli, which are, on that account, constant residents of stables. Sanchez and Veillon found them even in the dung of healthy horses and cattle ; and Sormani discovered them in the fæces of dogs, rabbits, guinea-pigs, and mice, and from these bacilli produced, by inoculation, tetanus in rabbits. The infection takes place most frequently by the penetration of earth-containing bacteria into wounds. Consequently, so-called traumatic tetanus is the ordinary form of the disease. The most frequent causes of tetanus are wounds from splinters of wood or gravel soiled with earth ; injuries of the feet by pricks in shoeing and gathered nails ; tread with subsequent soiling by dung, excrement or earth ; contused wounds which have come in contact with street dust ; and operation wounds, especially those of castration, performed without antiseptics. Tetanus may also be set up by infectious secretions of wounds, either by direct contact, or indirectly, by instruments, to which such secretions may adhere ; or by the falling of tetanus spores in the form of dust, on wounds. It has been clearly demonstrated that the dust of hay may be the bearer of tetanus bacilli. Evidently there are many other intermediate bearers. It has not yet been decided with certainty whether infection may take place through the intestines.

[I saw in Calcutta some years ago, several cases of tetanus which had been caused by gathered nails in horses that had been exercised on the Calcutta racecourse. This riding-track had at that time been laid down with horse-dung which had been obtained from ships that had brought large numbers of horses from Australia to Calcutta, and which contained as is usual in such cases, a plentiful supply of old nails. Consequently, this decomposing dung was a remarkably favourable means for the dissemination of the disease. The obvious lesson is, that when such manure is applied to ground on which horses are to be exercised, all incisive objects should as far as possible be removed from it.—TR.]

The views formerly held concerning the action of tetanus bacilli on the animal body were erroneous. It has now been proved that tetanus bacilli can alone, and not necessarily associated with the bacteria of putrefaction, of pus, etc., cause tetanus. The tetanus bacilli are however distinguished from most bacteria, particularly from those of septicæmia, by the fact that they do not penetrate into the blood and tissues of the body, but remain in the site of infection, from whence they poison the body by their products of metabolism (toxins and ptomaines).

Tetanus is therefore a toxic infective disease, in which a stationary vegetation of bacteria leads to a general intoxication of the body. This circumstance explains the observed fact that inoculation with the blood of animals suffering from tetanus always gives negative results, whilst inoculation with the bacteria-containing pus of the infected part produces positive results. The local colony of tetanus bacilli forms, during vegetation, chemical products (toxins and ptomaines) which have an action similar to that of strychnine, and which poison the body from the focus of infection. Brieger has demonstrated the existence, in tetanus cultures, of four strychnine-like toxins, namely, tetanin, tetanotoxin, spasmodoxin, and a hydrochloric acid toxin. These toxins are rendered ineffective by the gastric juice, intestinal epithelium, by many chemical agents, such as permanganate of potash, and by a temperature of 150° C. (Fermi). They are to be found in very great quantities in the tetanised muscles, in the blood and urine (Courmont), and in the milk (Brieger). Tetanotoxalbumin has lately been mentioned as the most important toxin.

The possibility of the transmission of tetanus from one animal to another, was demonstrated for the first time by the experiments of Carle and Rattoni in 1884. Recently, Kitt was the first to transmit pure cultivations to horses, sheep and dogs with the result of a typical tetanic affection.

**Occurrence.**—Tetanus occurs in all domestic mammaliæ, but most frequently in horses, asses, and mules; next to them, in the smaller ruminants (sheep and goats); and most rarely in dogs and birds. In the tropics and other hot climates, cases of tetanus including those of man are decidedly more numerous than in colder regions. For instance, Wagenfeld states that tetanus is so frequent in St. Domingo that a gelding costs twice as much as a stallion. In general, however, tetanus is a rare disease. In the Prussian army there is on an average only one

case of tetanus in 1,000 sick horses. For instance, 33 cases of tetanus occurred in 1890 among 36,000 sick horses. Within the last 7 years, only 270 horses (40 per annum) were attacked by tetanus in the Prussian army. According to Hering, 1 case of tetanus occurred in 3,000 cases of disease amongst the cavalry horses of Württemberg. Sometimes the disease breaks out with surprising frequency, indeed epizootically, among sheep and horses. Röhl and others have called attention to the fact that in veterinary hospitals tetanus is often not seen for months, or only very rarely in single instances; but that at other times, 2, 3 or more cases are brought in together. The occurrence of these temporary and local enzootics is explained by the infectious nature of the disease. Although a direct transmission of tetanus from diseased to healthy animals does not occur, it is nevertheless possible that a horse suffering from tetanus may infect the stable floor for a long time, so that the bacilli of tetanus may get from the floor into accidental wounds of healthy horses. Mestre saw 3 successive cases of tetanus in 3 horses which had occupied the same place in a stable.

Horses of high breed and strong constitution seem more susceptible to tetanus than underbred and less vigorous animals. A horse seldom gets tetanus twice (immunity). Nevertheless some horses have suffered from tetanus 2 or 3 times within a year. Among cattle, tetanus most frequently attacks cows and calves; among sheep, lambs; and among goats, bucks. Fowl are supposed to be immune.

**Varieties of Tetanus.**—Formerly 3 kinds of tetanus were recognised, namely, traumatic, rheumatic, and toxic tetanus. According to recent investigations we must reject the theory of the existence of rheumatic tetanus; for cold, like in other diseases, appears to play only the part of a predisposing agent.

Traumatic or infectious tetanus is the ordinary kind. Although experience teaches that any injury may be followed by tetanus; this disease develops by preference in contused and lacerated wounds, gun-shot wounds, punctures in which the foreign body remains, injuries of the nerves, tendons and fascia, wounds of the extremities in particular, and especially injuries of the feet of horses, contusions of the paws of dogs, and small and insignificant wounds. In horses, the chief sites of infection are wounds from gathered nails and pricks in shoeing; and to a lesser extent, wounds from tread, corns after having been cut



out, suppurating sand-cracks, and especially injuries to the sensitive sole. This predisposition of injuries of the hoof is explained by the theory of the wound becoming infected by dung. Spinola, very long ago, laid stress upon the danger of tetanus from infected wounds. It is probable that injuries of the hoof are much more frequently the cause of tetanus in horses than is usually supposed. Hartmann, for instance, showed that all the nine horses which were infected by tetanus in the course of several years at Dresden were found, on exact examination, to have suffered from hæmorrhagic pricks in shoeing, or injurious pressure from nails driven too "close." The fact is important that tetanus may occur even in wounds already healed and after the formation of a cicatrix (scar tetanus). Among other injuries conducive to tetanus are : saddle and harness galls, fracture of the vertebræ, fracture of the skull, burns, lacerations of the skin during petechial fever, penetration of foreign bodies into carious teeth, etc.

Tetanus may be observed after certain operations, especially castration. It has been proved by experience that nearly all the buck-goats which are castrated by certain methods, especially the ones by which the scrotum is opened with the knife, die of tetanus. For this reason, castration is now performed by ligaturing the entire scrotum, by including it between clams, or by the subcutaneous ligature of the spermatic cord. Although tetanus in rams may occur independently of castration ; in some years it appears enzootically or even epizootically as a consequence of that operation. Tetanus is liable to occur after the castration of bull-calves, especially when the scrotum has been ligatured, or when the spermatic cord has been subcutaneously ligatured. Besides castration, other operations—such as inserting a seton, applying a clam in cases of hernia, docking, nicking, pyro-puncture, subcutaneous injections, etc.—may be followed by tetanus, which, in such cases, was more common formerly than at present. The disease occasionally occurs in sheep after inoculation against variola and after the introduction of hellebore root into the nostrils. Since the introduction of antiseptics in operations, cases of tetanus have become rarer than they were ; which fact is a further proof of the infectious nature of the disease.

Internal wounds as well as external ones are frequently the cause of tetanus. Thus, cases of tetanus are not very rare in cattle after parturition or after abortion. It appears that the majority of cases of tetanus in cattle are caused during parturi-

tion by injuries to the uterus and vagina, difficult births, and putrescent decomposition of the after-birth, etc. Guibert is doubtless correct in saying that in cows, as in women, the obstetrician is sometimes the vehicle by which tetanus is transmitted from a diseased patient to a healthy one. Lesions of the intestinal mucous membrane, such as intestinal ulcers (Hering), may form the starting point of the disease.

In newly-born animals, especially in foals and lambs, the open wound at the navel may be the gate by which the infection of tetanus enters the body. In such cases, this disease may occur in lambs enzootically and epizootically (*Tetanus neonatorum*, and *tetanus agnorum*). Tetanus is also frequent among older foals.

It has been stated that tetanus can appear without any provable solution of continuity, as for instance by concussion, falls, etc. It is probable that in these cases the infection takes place through an injury which has been overlooked.

**Period of Incubation.**—The time tetanus takes to appear after an injury varies very much, being usually a few days. The shortest period on record for traumatic tetanus is 6 hours; the longest, 6 weeks. The time after parturition is, on an average, from 5 to 8 days; although it may vary from a few days to several weeks. In horses the period of incubation after inoculation is 4 or 5 days; and in sheep, 2 to 4 days (Schütz). Occasionally tetanus develops in spite of the wound having been treated antiseptically. The average period of incubation in horses may be taken as varying from 4 to 20 days.

**Toxic tetanus** has its cause most frequently in poisoning by strychnine, which is frequently observed in dogs. Brucin, nicotine, picrotoxine, thebain, caffein, ergotine, and other alkaloids also produce a tetanising effect. By the discovery of tetanotoxalbumins and other tetanising agents, traumatic tetanus appears to be exceedingly closely allied to toxic tetanus.

**Rheumatic tetanus**, according to the old view, developed after a rapid cooling of the skin, especially after sweating, by draughts of air, cold showers, remaining in the open during damp and cold nights, etc. It has been stated that the disease generally appears 2 or 3 days after the patient has taken a chill. In other cases there is a longer interval. For instance, Hamon reports that he observed tetanus 8 days after a cold brought on by a shower. In young animals, as in lambs, cold is often looked upon as the special cause of tetanus. Blame has often been laid on residence in stables and stalls which were situated on high and open ground; exposure when grazing during rainy weather; and chill after

washing the body. The existence of rheumatic tetanus has not been proved by exact investigations. In a case of rheumatic tetanus in man, Carbonne and Perrero found tetanus bacilli in the bronchial mucus.

**General Symptoms of Tetanus.**—The tetanic spasm begins usually in the muscles of the neck and head, and spreads from thence to the throat, trunk, and extremities (descending tetanus); but may appear first in the hind-quarters and extend forwards (ascending tetanus). In the former case, the symptoms commence with a stiff, stretched-out carriage of the head, and with spasms of the muscles of mastication. In the latter, with stiffness of the hind limbs and spasms of the tail muscles. The first symptoms are sometimes obscure, and may consequently be overlooked for several days. The disease may, however, break out suddenly with extensive tonic spasms. The spasms frequently begin near the site of the injury. The groups of muscles which become attacked by the spasms are as follows:—

1. *The muscles of mastication.*—The tonic contraction of these muscles is called trismus, or lockjaw. According to the degree of the spasm, the lower jaw remains in more or less close contact with the upper jaw, and consequently prehension of food and mastication are either rendered very difficult, or are entirely stopped.

2. *The other muscles of the head* are spasmodically contracted in different degrees. The spasm of the muscles of the ears causes the ears to be “pricked” and their tips drawn together; spasm of the recti muscles of the eyes induces retraction of the eyes into the orbit, with protrusion of the nictitating membrane; spasm of the muscles of the nose produces dilatation of the nostrils; and contraction of the dilators of the upper lip gives an abnormal shape to the opening of the mouth. The muscles of the tongue, of the swallowing apparatus, of deglutition, and of the larynx, are also attacked by spasm.

3. The contraction of the *extensor muscles of the neck* causes a stiff, stretched-out carriage of the head and “ewe neck.” The muscles of the neck become hard and tense to the touch.

4. Spasm of the *extensor muscles of the back* manifests itself by an extremely hard condition of the muscles of the back, loins, and croup. Either the neck is stretched out and the back and croup are carried horizontally (*orthotonus*), or, what is more frequently the case, the neck is “ewed” (head raised and drawn back) and the vertebral column slightly depressed (*opisthotonus*). A lateral curvature of the cervical vertebræ (*pleurothotonus*) is not so often seen as the previously mentioned conditions; and

a convex curvature of the vertebræ (*emprosthotonus*) is rarest of all. The tail, especially in horses, is raised and frequently forms a straight line with the back.

5. Spasm of the *muscles of the limbs* makes the animal assume an attitude in which the fore and hind legs are stiffly stretched out laterally, and respectively, to the front and rear, like those of a trestle. The legs can be bent only with difficulty, or not at all.

6. The contraction of the *muscles which compress the abdomen* gives the animal a tucked-up or "herring-gutted" appearance.

7. The spasm of the *muscles of inspiration* renders respiration difficult.

Besides these spasms, the animals show, in various degrees, heightened sensibility and *increased reflex irritability*, which manifest themselves in excitement, timidity, and exacerbations of the muscular spasms; although the amount of the irritation may be very slight. Thus, for instance, a loud noise, as the banging of a door, may cause the animal to fall down "all of a heap." On the skin we generally find *sweat*, which, in severe cases and towards death, is very abundant and is diffused over the whole body. In other and especially in slight cases, sweat may be absent. Usually the internal temperature is but little if at all increased. Only in fatal cases is the elevation of temperature constant, though often it is very high ( $42^{\circ}$  or even  $43^{\circ}$  C.), but only for a short time before death. This high temperature usually continues some time after death and may then rise to  $45^{\circ}$  C., or even more. In a horse, 24 hours before death, Bayer observed a temperature of  $39.2^{\circ}$  C.; one and a half hours before death,  $41.2^{\circ}$  C.; at the moment of death,  $44^{\circ}$  C.; and 50 minutes after death,  $45.4^{\circ}$  C. The frequency of the pulse is not increased at the commencement of the affection. In slight cases the pulse may remain normal, which it generally does, with only a trifling increase in frequency, until severe exacerbations set in. In many cases, the rate of the pulse rises only during the last few days, and may then be from 70 to 90 beats *per* minute in the horse. The frequency of the pulse of animals which continue recumbent, is much greater than that of those which keep on their legs. The pulse is often hard and small, and the wall of the artery spasmodically tense. In many cases the pulse is full, soft, and easily compressible.

Increase in the *rate of breathing* is shown generally at the commencement of the disease, and may become very high when the spasms spread to the muscles of the chest and abdomen. The

rate of respiration varies greatly according to the excited condition of the animal. As a rule, it gradually increases in cases which end fatally. It may amount to four times its normal standard, without the frequency of the pulse being increased to any marked extent. When the affection is at its height, the number of respirations in horses rises sometimes to from 80 to 100 a minute. Respiration is usually more or less shallow, on account of the fixed condition of the ribs, and the restriction to the movements of the diaphragm in consequence of the spasm of the muscles which compress the abdomen. We may also notice cyanosis and catarrh of the nasal mucous membrane, coughing, laryngeal, tracheal and bronchial rhonchi; and in fatal cases, symptoms of hyperæmia of the lungs, œdema of the lungs, hypostatic inflammation of the lungs, and often pneumonia caused by foreign bodies.

The first indication of the participation of the *digestive apparatus* in this disease, is afforded by its action on the prehension of food, which is difficult or even impossible, although the appetite may be unimpaired. Some patients can eat comparatively easily; others, only with much trouble and after great exertion, can manage to consume a portion of their food, which frequently accumulates in the oral cavity, and becomes putrid or falls out. Considerable salivation is present. The slight movements of mastication, especially in horses, are accompanied by squashing sounds and smacking of the lips. The animal can generally partake of sloppy food easier than dry. Regurgitation is very common in consequence of spasm of the pharyngeal muscles. The patients are always fond of playing with the drink placed before them, and make frequent, although very often futile attempts to satisfy their thirst, which increases during the course of the disease.

As a rule, the *peristaltic action of the bowels* is somewhat suppressed, and defecation is always delayed on account of the rigid contraction of the muscles which compress the abdomen. Micturition becomes less frequent and more difficult, even to complete retention of urine, in consequence of which the bladder becomes excessively dilated (spasm of the sphincter vesicæ). The urine has generally a rather high specific gravity and sometimes contains albumen. Its reaction is usually not changed. Frequently occurring and long continued priapism may be noticed in stallions, and even in geldings.

Consciousness is perfectly preserved with all animals close up to death; although the feeling of terror is usually mani-

fested in a very unmistakable manner. The state of the infected animals arouses in the spectator, and especially in the surgeon who treats them, deep compassion for their helpless condition.

#### TETANUS IN PARTICULAR KINDS OF DOMESTIC ANIMALS.

**The Horse**, in the fully-developed stages of the disease, stands with the head raised and the neck stiffly stretched out, and sometimes "ewed," especially in foals. As we have already said, the ears are stiffly pricked and approach one another, reminding us of the ears of hares; the nostrils are rigid and dilated into the form of trumpets; the eyes are sunken; the nictitating membrane protudes; the pupils are dilated; the veins of the head are tense and prominent; foam and saliva run from the mouth; the tail is raised, and usually carried a little to one side. To enlarge the area of his base of support, the animal assumes the trestle attitude (spreads his legs out), and does not change it, or, at most, now and then rocks his body to and fro on his stiff legs. The appearance of the face betrays marked anxiety and excitement. Several groups of the muscles, especially the masseter and cervical muscles, are remarkably dry, prominent, sharply defined, as if they had been chiselled out, and evidently in a state of excessive tension. Sweat may be seen on several parts of the body, especially at the base of the ears, sides of the neck and chest, and flanks, which are greatly tucked up. Respiration is difficult and accelerated. If we approach the animal we cannot fail to note the anxiety and terror which it evinces on being touched, and especially if its head be handled. The wings of the nostrils, external muscles of mastication, and muscles of the neck, back, loins, croup and tail, are tense and as hard as a board to the touch. It is difficult to alter the position respectively assumed by the tail and ears, which resume it more or less rapidly on the restraint being removed. All the visible mucous membranes of the head appear congested. The jaws are firmly closed and cannot be forced open. When, for this purpose, the fingers are introduced between the bars of the mouth, considerable quantities of tenacious mucus, or turbid, whey-like foetid fluid, which is mixed with particles of food, flows from the mouth. On the head being raised, the haw becomes visible to its full extent, so that it frequently covers the greater part of the eyeball. If we attempt to back the animal out of its stall, the movement to the rear is accomplished only with the greatest difficulty or not at all. The horse turns with extreme difficulty without bending the trunk and neck, and only slightly flexes and raises his legs, which he uses like stilts. The gait, which may gradually improve a little, is exceedingly stiff and straddling.

In **cattle** the symptoms of tetanus are frequently modified by the disease being complicated with acute or chronic septic endometritis. The animals assume, with outstretched limbs, the trestle-like, rigid attitude already described. The head and neck are stretched out; the ears are stiff and directed backwards, upwards and outwards; the expression of the face is staring or anxious; the eyes are deeply retracted into their cavities; the nictitating membrane, as in the horse, covers the greater part of the eyeball, especially when the head is raised; the muzzle is sometimes dry and warm; and the mucous membrane of the mouth is

hot, and secretes quantities of tough foetid mucus. The lips are somewhat drawn back; the mouth is spasmodically closed; the lower jaw is firmly fixed to the upper one; the muscles of mastication are tense and hard as a board; the tongue possesses little mobility, is hard and sometimes firmly fixed between the jaws; the muscles of the throat, back and extremities are hard to the touch; the spinal column is sometimes arched (*emprosthotonus*); and the abdominal walls are at times drawn in laterally in a peculiar manner, so that they form a flat surface downwards, from the ends of the transverse processes of the lumbar vertebræ. Great difficulty in breathing is met with only towards the termination of the disease. In consequence of the suppression of the movements of the paunch, tympany of the left flank frequently appears in a few days. Defecation and micturition are delayed. The locomotory movements are stiff, awkward, and often entirely suppressed. The hocks are sometimes rotated outwards, and the tail raised. Reflex irritability is usually less altered than in the horse.

In **sheep** the hind-legs, and, later on, the fore-legs, are stiffly stretched out, so that the animal generally rests immovable as if on four stilts. The tail is rigid, carried straight out, and is frequently deflected somewhat sideways. The neck is stiff and "ewed," and the jaws closed. Later on, the animal drops down and lies with its legs extended straight out, the neck being drawn backwards, on which account respiration is rendered excessively difficult. In other particulars, the symptoms of the disease agree with those in horses and cattle. From existing literature it is difficult to obtain an exact description of tetanus in lambs; for tetanus in them has frequently been mistaken for other diseases, especially pyæmic polyarthritis. The symptoms of tetanus in **goats** are the same in every respect as those in sheep.

General tetanus appears to occur very rarely in **dogs**. Möller found among 50,000 diseased dogs, only 2 cases of general tetanus. We ourselves failed to discover a single case amongst 70,000. Partial tetanus in the form of trismus seems to occur sometimes in puppies. Recent inoculation experiments prove that dogs are not absolutely immune to tetanus, as was formerly supposed. The symptoms of general tetanus consist in a stiff, stilty gait; extended attitude of the head; elevation of the neck; the ears are stiffly pricked, and sometimes laid back; staring, anxious expression of face; protrusion of the nictitating membrane; corrugation of the skin of the forehead; trismus, which renders the patient incapable of barking; extension of the muscles of the neck ("ewe neck"); stretched-out and stilty attitude of the legs; orthotonus; opisthotonus; erection of the tail; excessively increased reflex irritability, especially on being suddenly touched; etc.

In **pigs** the symptoms of tetanus are essentially the same as in other animals. The tonic spasm usually begins in the muscles of mastication and spreads rapidly, as a rule, over the entire body (trismus, orthotonus, and emprosthotonus).

In **birds**, Dreymann has described one case, namely, that of a turkey. The gait was stiff, the head and neck were extended, and the entire muscular system was stiff and hard. The beak could be opened only with difficulty, and finally became firmly closed. The wings lay close to the body. The membrana nictitans was protruded, and dyspnoea was present.

**Pathological Anatomy.**—The results of a *post mortem* examination of a case of tetanus are usually negative. Similar to what is the case in narcotic poisoning, there are no characteristic anatomical changes. Any changes that may be found are only secondary, or are set up by accidental complications. Thus we may sometimes find in the spinal cord, hæmorrhages, softening, accumulation of serum, hyperæmia, œdema, etc., which changes, however, may be entirely absent, or only secondary, and are frequently traumatic. We may find in the course of the nerves which proceed from the wounded parts, in some places, a pink injection of the nerve sheaths, very minute hæmorrhages, and swelling or softening of the nerve fasciculi (*neuritis ascendens*). On account of impeded oxygenation, the blood is usually of a dark-red colour, tarry and greasy appearance, is greatly discoloured, and is imperfectly coagulated. It contains free hæmoglobin, and is inclined to escape from its vessels and to become absorbed into the surrounding tissues. We may observe numerous extensive ecchymoses, and large sanious and gelatinous extravasations, especially under the serous and mucous membranes. The lungs are generally hyperæmic or œdematous, and hæmorrhagic infarcts, splenisations, hypostatic pneumonia, lobular, gangrenous pneumonia, and vesicular and interstitial emphysema, may also be present in some parts. In the heart we find epicardial and endocardial hæmorrhages, particularly along the furrows of the blood-vessels and over the papillary muscles, and fatty degeneration of the myocardium. Rigor mortis, which is generally highly developed, appears rapidly in the muscles, which may be in a normal condition, or may be of a dull, dark-red colour, and infiltrated with hæmorrhages. On other occasions it is bluish-brown, or yellow-brown, and soft as if boiled. In the muscles, we can sometimes find, by the microscope, a granular cloudy swelling, with loss of transverse striation and splitting up of the muscular fibres in layers. The liver is usually enlarged and is frequently of a yellow or yellow-grey colour (fatty degeneration and infiltration). The spleen is swollen, gorged with blood, and flabby. The bladder is usually considerably distended by the accumulated urine, and its mucous membrane is often infiltrated with hæmorrhages. Ecchymoses are not uncommonly met with on the mucous membrane of the intestines.

**Prognosis.**—The prognosis of tetanus is usually very unfavourable. The mortality amounts in sheep and pigs almost



to 100 per cent. ; in horses from 75 to 85 per cent. ; and in cattle from 70 to 80 per cent. In dogs it is comparatively small. Out of 270 horses which were treated for tetanus during the years 1887-1893 in the Prussian army, 203 died. In newly-born animals the disease is usually fatal, especially in lambs. In cattle, the prognosis of a case of tetanus following parturition is less hopeful than of one of ordinary tetanus. The chief unfavourable symptoms are : early and excessive trismus (horses in which trismus is not followed by general tetanus mostly recover) ; rapid spreading of the spasms over the entire muscular system ; excessive dyspnoea ; rapid and considerable increase in the rate of the pulse and in the height of the temperature ; falling down ; profuse sweating, etc. The fact of the disease running a protracted course is favourable. With horses we may hope for recovery when the patient lives beyond the fifteenth day of the disease.

Schindelka observed 4 cases of tetanus in which a complication with influenza had a peculiarly favourable influence. In all 4 cases the muscular rigidity abated simultaneously with the commencement of the first feverish symptoms, and recovery followed. Bassi states that a similar effect was produced by parturition in a mare.

**Course.**—The course of tetanus is variable. In horses, it is frequently very brief, and then ends fatally in 1, 2, or 3 days. Recovery in rapidly developed cases has never been observed. The animal exhibits at an early period of the disease, excessive trismus, spasms over the whole body, dyspnoea, greatly increased reflex irritability, profuse sweating, considerable acceleration of the pulse, and increase of temperature. If the horse falls down, it will die as a rule very quickly. Usually death takes place *in about a week's time*. A fatal course of tetanus may run for 2 or 3 weeks, or even more, if the spasms are slight and more or less local, or when, after a cure, complications, such as pneumonia due to a foreign body, set in. Thus, Köhne had a case of a horse which died from exhaustion and cavities in the lungs on the fifty-seventh day. Recoveries rarely take place earlier than 3 weeks, about which time the spasms usually abate in a gradual manner ; the appetite improves ; respiration becomes more tranquil ; the movements become freer ; and the animal becomes convalescent in 5 or 6 weeks ; although stiffness of gait and considerable tension of the muscles of the spine may persist for a

much longer period. Convalescence is generally prolonged for some months.

In *cattle* the course is less rapid than in horses. The spasms take longer to become developed, and are not, as a rule, so intense. In exceptional instances, even 10 days or a fortnight may pass before complete trismus sets in. Acute cases which end fatally in a few days may also occur in cattle. Recovery does not take place in less than a fortnight. Contrary to what we find in horses, the disease seldom lasts longer than 3 weeks.

In *sheep*, death generally occurs in about a week ; and in acute cases, which are not rare, in 2 or 3 days. The course of the disease may be equally rapid in *dogs*, especially in cases of traumatic tetanus ; but in other cases it may end fatally in from 6 to 10 days. Under favourable circumstances, recovery, as a rule, takes place in 12 or 14 days.

**Differential Diagnosis.**—Tetanus may be mistaken for a number of diseases, as for instance, cerebro-spinal meningitis, rheumatism, eclampsia, catalepsy, rabies, convulsions in newly-born animals, and pyæmic polyarthritides in lambs and foals. The differentiation, as a rule, is not difficult. The continued tonic spasms of the different groups of muscles, the perfectly preserved consciousness, the absence, at first, of fever, and the characteristic aspect, render diagnosis easy. It may, however, be difficult to distinguish infectious tetanus from poisoning by strychnine. The extreme rarity of infectious tetanus in dogs, the suddenness of the attack, the rapidity of the course, and the greatly increased reflex irritability in strychnine poisoning, are valuable diagnostic facts.

**Therapeutics.**—Our own experience and that of many others teaches us that in the treatment of tetanus the administration of medicine is of much less use than careful nursing, of which the most important points are appropriate dieting and the avoidance, as much as possible, of excitement from external causes of irritation. We should place the patient in a quiet, dark, and sufficiently large stall or loose box, and well bedded-down, in view of the possibility of his falling on the ground all of a heap. If the straw used for litter be too long, it should be cut in two, so that the patient may not catch his feet in it and fall down. Horses should, if possible, be placed in slings, which we, contrary to the experience of other observers, have always found

useful, when we have been able to use them. The excitability of the animal, which is perhaps at first somewhat increased by the employment of slings, soon abates. The great advantage of slings lies in the fact that they safeguard the patient from unexpectedly falling down, especially during the night, and thus obviate the possibility of prolonged continuation of the recumbent position, which is always followed by very severe exacerbations of the disease. Feeding is a very important question. If dry food cannot be eaten, we should place before the animal, if possible, green meat, sloppy food, such as gruel, and above all things, fresh water. The food should be given frequently, and in small quantities at a time, so that it may not become spoiled, which slops, gruel and bran mashes, very soon become. When perspiration is excessive, the clothing should be frequently changed. Dressing the coat, which is approved of by many, should be avoided, on account of the excitement it causes to the patient. If retention of fæces and urine persists, we should, in the larger animals, back-rake the patient from time to time, and should apply slight pressure on the bladder with the hand. We may use the catheter in females; and enemas with the smaller animals.

As regards medicinal remedies, we may exhibit narcotics tentatively, as for instance, enemas of chloral hydrate, 50 to 100 grammes or more, *pro die*, in two or more doses, or, say, 10 grammes every hour, in thin mucilage for horses; subcutaneous injections of morphia ( $\frac{1}{2}$  gramme of the hydrochlorate for horses); inhalations of chloroform; injections of ether into the rectum (25 to 50 grammes *pro dosi*). Medicines should not be administered *per os* on account of the danger of trismus being set up, and of the liability of the drench or ball going the "wrong way."

In cases of traumatic tetanus, it is of the utmost importance to apply as soon as possible appropriate treatment to the wound by disinfecting it, removing from it penetrating foreign bodies, excising or cauterising the infected part, or even performing amputation. Animals can be undoubtedly saved by this local treatment, if it be early employed and if it be practicable. We must, however, remember that an exact examination and special treatment of the wounds in the larger animals may be attended by considerable difficulty. With injuries of the feet of horses, it is frequently nearly impossible to raise the leg off the ground without incurring the risk of causing the animal to fall down. If, in such a case, the condition of the wound urgently requires

local treatment, nothing is left to us but to place the horse under the influence of chloroform, by the aid of which we may perform the necessary operation. We have not sufficient *data* at present to pass a final judgment on the value of neurotomy in traumatic tetanus. Rosco states that plantar neurotomy was followed by rapid improvement and cure in a case of tetanus caused by a gathered nail.

As *prophylactic treatment*, we recommend the careful disinfection of the place of residence of animals suffering from tetanus. Clinical experience has taught us that the stalls and loose boxes of infected horses continue, for several years, to be dangerous centres of infection (Hartenstein).

**Immunity against Tetanus.**—Behring and Kitasato have experimentally proved that animals may be rendered immune to tetanus by successive attenuated pure cultivations of tetanus bacilli which have been suitably treated with iodine trichloride. This artificially-produced immunity depends on the fact that cell-free blood serum of these animals possesses the power to render inert the toxin produced by the tetanus bacilli. The system of the animal which has been protected by inoculation, consequently contains an effective chemical substance, namely, an antitoxin, on the presence of which, in the blood, the power of immunity depends. Inoculations with the blood serum of animals which have thus been rendered immune to tetanus, are supposed to confer on other animals immunity against subsequent infection with tetanus toxin, or infection with living tetanus bacilli. Behring made these experiments with small animals, such as rabbits, guinea-pigs, and mice. Schütz, Koçh, Behring, and Kitasato carried out this method of obtaining immunity in horses and sheep. The animals experimented upon were repeatedly injected subcutaneously with different quantities of cultivations of tetanus bacilli, which had been mixed with iodine trichloride and had thus become weakened. The proportion of iodine trichloride, which at first was from half to one per cent. was gradually reduced to a quarter and then to an eighth per cent., so that the cultivations used for the immunising injections were of increasing virulence. This treatment, which was continued for about a month with intervals of several days, rendered the experimental animals immune to even fully lethal cultures of tetanus bacilli, which proved fatal to the unprotected control animals. From these experiments Schütz arrives at the following conclusions :—"Horses and cattle can by Behring's method be protected not only against an infection with living tetanus bacilli, but also against the injurious effects of the virulent substances which are formed by the tetanus bacilli in cultivations or in the animal body. The power of resistance of horses and sheep which have been rendered immune to living tetanus bacilli and to the specific virus of tetanus, becomes increased by continued subcutaneous injections of cultivations of gradually increasing strength, or of gradually increasing quantities. The blood of these animals acquires immunising properties, which increase proportionately to the increase in the power of resistance."

Behring's therapeutic experiments with the tetanus immune serum gave negative results, in that it produced no curative effect on animals suffering from tetanus, as was proved by the experiments made in the Prussian army.

### GLANDERS (*Equinia*, *Malleus*).

Glanders in horses—Glanders in cats and beasts of prey—Glanders in man—  
Lymphangitis epizootica.

**History.**—Glanders (glanders and farcy) is one of the oldest known diseases of horses. Aristotle and Hippocrates were acquainted with farcy, and glanders of the donkey. The Roman authors, Apsyrtus and Vegetius, have described glanders, and Vegetius recognised several kinds of glanders ("*malleus*"), especially that of the nose and skin ("*malleus humidus et farciminosus*"). In the Middle Ages, glanders was considered, by the German laws, to be a legal unsoundness (breach of warranty). The infectiousness of glanders was recognised as early as the seventeenth century. Solleysel (1664) supposed that it could be transmitted by the air. Van Helmont (1682) considered it to be identical with human syphilis. Even in 1734, Gaspard Saunier gave precise directions about disinfecting stables. Garsault (1741) and Bourgelat (1764) recommended immediate slaughter of horses suffering from glanders and segregation of suspected animals. The identity of glanders and farcy was clearly recognised at that time. At the end of the eighteenth century, Abildgaard and Viborg in Denmark proved by a great number of experiments that glanders could be transmitted from one horse to another. These investigators demonstrated, at this early period, that the virus of glanders was fixed; that it could be transmitted by the respiratory air; that the transfusion of the blood of horses suffering from glanders is far less efficacious for transmitting the disease than inoculations of secretions or of pus; and that the virus loses its power of infection if dried or heated to 45° C. Similar experiments were made in England by Coleman and Delabère Blaine, who recognised that the nasal discharge was the principal vehicle of the virus.

On the other hand, the theory of the contagiousness of glanders was much doubted at the beginning of last century. Especially in France, the view taken by the Alfort Veterinary College, that glanders might arise spontaneously from an attack of strangles, was far more widely accepted than the doctrine of its contagiousness, which was stoutly supported by the Veteri-

nary College of Lyons; the consequence being, that precautionary regulations against glanders were abolished, with the result that the spread of the disease increased to an extraordinary extent in France. At that time, glanders was looked upon as tuberculosis (Dupuy), or as simple pyæmia (Vatel and Bouley). It was only when Rayer (1837) had proved the transmissibility of glanders to man, and when Chauveau (1868) had shown that the virus was contained chiefly in the firm component parts of the infective material, that the fact of the contagiousness of the disease was again accepted.

The theory of the spontaneous origin of glanders was accepted by every one in Germany towards the middle of last century. It was believed that glanders could be produced by the injection of pus; and that strangles could turn to glanders (Hering and Funke). Glanders was looked upon, respectively, as a tubercular disease, scrofula (Haubner), pyæmia, diphtheritis, general dyscrasia, and cachexia. Virchow was the first to declare that the nodules of glanders were independent anatomical formations, which he placed under the heading of granulation tumours. Gerlach was the great advocate for the exclusively contagious origin of glanders. He threw much light on the clinical and diagnostic aspects of the disease. We are indebted to Leisering for the first exact anatomical description of glanders.

The first bacteriological researches were made in 1868 by Zürn and Hallier, who found a fungus which they believed to be the cause of glanders, and to be identical with that of human syphilis. In the same year, Christot and Kiener thought they had discovered the micro-organism of glanders. Bouchard, Capitan, and Charrin, in 1881-82, sought to isolate the virus of glanders. It was not, however, until the year 1882 that Löffler and Schütz succeeded in finding the true bacillus of glanders, in cultivating it, and in transmitting it to other animals. Their researches have furnished the positive proof that glanders is a specific infectious disease, produced exclusively by the bacillus mallei.

**Bacteriology.**—According to Löffler and Schütz, the bacilli of glanders are rods, the length of which is from 1 to 3  $\mu$ , or from one-third to two-thirds the diameter of a leucocyte. They are from 5 to 8 times as long as they are thick, are straight or slightly curved, and have rounded ends. They are somewhat thicker and shorter than tubercular bacilli, and are generally found in couples connected together longitudinally. They stain

in solutions of methylene blue. The cultures on gelatine appear on the surface of the gelatine, as small, limpid, yellow, translucent drops, and assume, later on, a milky-white colour. The potato cultures, which are very characteristic, form after the third day a uniform, amber-coloured, translucent covering, which assumes in about a week a reddish tinge similar to that of sub-oxide of copper. A greenish hue appears in the neighbourhood of the cultures. The bacillus grows best on solid blood serum of horses and sheep; on neutral broth made of the flesh of horses, cattle, fowls, and human beings; and on slices of potato. The temperature limits of the growth of the fungus are  $20^{\circ}$  C. and  $45^{\circ}$  C. Consequently, summer heat favours its growth, which is impeded or destroyed by temperatures under  $20^{\circ}$  C. and over  $45^{\circ}$  C. For this reason it is impossible that an ectogenous development of the fungus can take place. Löffler failed to obtain cultures on infusions of hay, straw, or horse-dung. Glanders appears therefore to be a purely contagious, and not a miasmatic or miasmatic-contagious affection. As a rule, complete desiccation of the bacilli of glanders destroys their virulence in about a week. According to the experiments of Löffler, a period of 3 months is the longest time the dried bacilli retain their activity. Cadéac and Malet state that the bacilli can be killed only by gradual drying; that they resist putrefaction from 14 to 24 days; and that, when mixed with water, they continue virulent from 15 to 20 days. Bacilli which are not dried, cannot live outside an animal's body for longer than 4 months. Löffler therefore considers that 4 months is the maximum period for the infectious material to retain its virulence, and that the published reports about stables remaining infectious for many months and even years, are erroneous. Löffler regards the formation of spores to be very doubtful on account of the slight power of resistance possessed by the bacilli. Rosenthal, however, treated glanders bacilli taken from old potato cultures after Neisser's method of staining spores, and obtained exactly similar forms as with the spore containing bacilli of anthrax. We must therefore admit the possibility of spore-formation.

Many contradictory statements have been made about the power the bacilli possess of resisting the action of disinfectants. Some authorities state that an exposure to a temperature of  $55^{\circ}$  C. kills them in 10 minutes, or of  $80^{\circ}$  C., in 5 minutes; and that they die if left for 5 minutes in a 3 to 5 per cent. solution of carbolic acid or creolin, or for 2 minutes in a watery solution of

chlorine, a 1 per cent. solution of permanganate of potash, or in a one-fiftieth per cent. solution of corrosive sublimate, etc. The experiments of others show that the bacilli are not always destroyed by an exposure of half-an-hour to a temperature of 100° C. (Bromberg). For practical purposes we may say that a 1 to 1,000 solution of corrosive sublimate, or a 5 per cent. solution of creolin or carbolic acid, is sufficient for disinfection. Iodoform considerably impedes the development of the bacilli outside the animal body, so that their infectiousness is lost when they have been for some time under its influence. Inside the animal body, iodoform impedes but little the development or the dissemination of the bacilli. According to the experiments of Gamaleïa, the virulence of the bacilli of glanders may be increased by passing it through the body of the earless marmot [*spermophilus citillus*], or rabbit. Although the digestive fluids weaken fresh cultures of the bacilli of glanders, they do not kill them (Mozarski).

**Occurrence.**—Glanders is essentially an equine disease and affects donkeys, mules and jennets, as well as horses. It is transmitted from the horse to many other animals, including man, either in the natural course of events, or by experimental infection. The order of comparative susceptibility to the virus of glanders possessed by certain animals is somewhat as follows:—Field-mice and guinea-pigs; the cat tribe, including lions and tigers; dogs, goats, rabbits, and sheep. Pigs and pigeons are scarcely at all susceptible. Cattle seem to be quite immune. In fact, there has never been a single well-authenticated case of glanders observed in them. The long-established practice of isolating glandered horses in cowsheds has never produced any bad effect on the cattle residing in these abodes. House-mice, white mice, rats, domestic fowl, and linnets are immune.

There is a great difference in the individual susceptibility of horses to the virus of glanders, which some readily take, while others may remain for months and even years in a stable with glandered animals without becoming infected. Glanders, like tuberculosis, has certain predisposing factors, the chief of which are: over-exertion, too little food, faulty stable management, defective ventilation, chill, and disease. The spread of glanders is therefore greatest during times of war.

Although glanders is far less frequent at present among horses than formerly, thanks to veterinary police laws, it still



continues to be the most dangerous equine disease. It occurs in all latitudes and in all climates.

**Statistics.**—In the German Empire, official notification was given, during the 9 years from 1886 to 1894, of 8,717 case of equine glanders, which is an average of nearly 1,000 cases per annum. The respective years show the following numbers:—1,220 (1886); 1,228 (1887); 1,182 (1888); 1,337 (1889); 866 (1890); 981 (1891); 823 (1892); 564 (1893); and 516 (1894). According to these figures, we see that there has been a steady decrease in this disease. The average percentage at present in Germany is from 0.02 to 0.03 of all the horses.

2. Felisch computes that from 1876 to 1886, 20,566 horses in Prussia died of glanders or were killed on account of having glanders, and gives the following figures for the respective years:—2,740 (1877); 2,848 (1878); 1,994 (1879); 2,182 (1880); 2,357 (1881); 2,297 (1882); 1,568 (1883); 1,879 (1884); 1,525 (1885); and 1,176 (1886). According to this, the losses diminished considerably more than one-half during these ten years. The decrease began with the introduction of the Imperial laws for the regulation of epizootics. The principal losses were in Posen (0.22 per cent.), West Prussia (0.15 per cent.), and in Silesia (0.13 per cent.). The disease was most rife in the districts of Oppeln, Bromberg, Posen, Marienwerder, and Dantsic (Russian and Polish-Russian frontier). Draught animals formed 22 per cent. of the glandered horses. There were 20 known cases (3 of veterinary surgeons) of the disease having been transmitted to man. The total sum paid as indemnity amounted to £212,500.

3. According to Röll, there were 3,317 horses (0.3 per cent. of the entire number) officially reported as being glandered in Austria during the years 1877 to 1887.

4. In Hungary, 1,661 horses became glandered in 1891 and 1892.

5. In France about as many horses as in Germany (5,623) became infected from 1886 to 1890.

6. The number in Great Britain\* during the same period amounted to 8,000. In 1890 there were 782 cases of glanders in London alone.

7. According to a table compiled by Krabbe for a period from 1857 to 1873, there were yearly among 100,000 horses in Norway 6 cases of glanders; in Denmark, 8.5; Great Britain, 14; Württemberg, 77;

\* According to the official report, the respective numbers of horses which have been attacked by glanders during the years 1895 to 1901 in England are as follows: In 1895, 487 cases; in 1896, 1,196; in 1897, 1,324; in 1898, 1,133; in 1899, 1,269; in 1900, 1,814; and in 1901, 2,304. The measures adopted by government for the suppression of glanders are inadequate, and have been carried out in an inefficient manner. London and Glasgow are the two great centres of glanders in Great Britain. During the last few years, there has been a well marked increase of cases of glanders in London. Formerly, glanders was a common disease among our army horses; but, thanks to the vigilance displayed by our military veterinary authorities, there was not a single case of glanders among army horses in Great Britain from October, 1888, up to the Boer war, during which period, glanders spread to a comparatively large extent among our army horses.—Tr.

Prussia, 78 ; Servia, 95 ; Belgium, 138 ; France, 1,130 ; and in Algiers, 1,548. It would be difficult to decide whether this increase of glanders from north to south, as Löffler assumes, is due to greater warmth of climate (the bacillus of glanders, as we have already said, thrives only at a temperature of over 20° C.), or to insufficient veterinary police regulations in the countries in question.

8. During the 10 years from 1877 to 1887, there were 4 cases of glanders each year among 100,000 horses in Sweden (Lindqvist).

9. Woronzow estimates the number of glandered horses in European Russia at 90,000, that is, 4 *pro mile*.

**Pathogenesis.**—The transmission of glanders from an infected animal may take place directly or indirectly through extremely different intermediate bearers, such as harness, clothing, pails, stable tools, fodder, litter, grooms, and other agents. The respective discharges from the cutaneous ulcers and from the nostrils possess the highest degree of infectiousness. The bacilli of glanders may occur in any organ as well as in the blood, which does not always contain them, except when the infection is general throughout the system. Cadéac, Malet, and others frequently obtained positive results from inoculation experiments with the blood of glandered horses.

The *respiratory organs* are most frequently (in nine-tenths of all cases) the gate of entrance for the virus. The bacilli, like those of tuberculosis, probably reach the nasal mucous membrane by being inhaled along with the air in the form of dry dust ; by the reciprocal smelling of neighbouring animals ; and by deep respiratory movements, as for instance, during severe harness work. We may here point out that the presence of cartarrh predisposes the respiratory mucous membrane to the penetration of the bacilli. According to the experiments of Renault and others, it seems to be very doubtful that the bacilli of glanders can be directly exhaled into the atmosphere and thus brought into contact with the air passages of healthy animals by inspiration. It is much more reasonable to assume that the transmission of the bacilli takes place by the bacilli-laden nasal discharge flowing out or being blown out, and by its becoming dried and then inhaled in the form of dust. Experiments to transmit the disease by the direct inhalation of the respiratory air of glandered horses have always been without result. It still remains an open question whether or not the nasal mucous membrane must be morbidly affected before it can successfully receive the infection of glanders ; although an affirmative answer has apparently been given by the experiments of Cadéac and Malet. It is probable that the bacilli

of glanders, like those of tuberculosis, can penetrate through the uninjured mucous membrane. They may also be introduced along with the inspired air into the lungs, where they then lead to the development of very minute bronchial-pneumonic foci. Glanders in the lungs, like glanders of the air passages of the head, may occur primarily. In fact it frequently happens that the lungs alone are affected, or that they show the oldest changes. Analogous conditions are present in tuberculosis and actinomycosis. For these reasons most authors believe in the possibility of a primary affection of the lungs in glanders (Leisering, Gerlach, Bollinger, Roloff, Pflug, Rabe, Röhl, Csokor, and Johne). At the same time, it cannot be denied that some cases of apparent primary glanders of the lungs are in reality secondary processes which had formed, by embolism, from the glandered ulcers of the nasal mucous membrane, after the primary foci had healed and had consequently escaped notice during examination. It is well known that the fresh ulcers in the nostrils may arise secondarily from old glanderous deposits in the lungs, from which the bacilli are carried with the expired air to the nasal mucous membrane. Even the acceptance of these cases cannot justify us in denying the possibility of primary glanders in the lungs.

The *skin* is a second gate of entrance for the bacilli of glanders ; farcy, which was formerly regarded as a separate disease, being nothing else than glanders of the skin. A primary infection of the skin is, as a rule, possible only when the skin has been previously injured. Cases of primary glanders of the skin are consequently uncommon. Far more frequently the skin becomes infected secondarily as a result of general glanders set up by emboli. Contrary to the case reported by Babes, Nocard failed to make the bacilli of glanders penetrate the uninjured skin of donkeys and guinea-pigs, by the inunction of a bacilli-containing ointment. If infection takes place after such an application, we must assume either the presence of an abrasion of the skin, or that the continuity of the skin has in some manner been injuriously affected by the friction employed.

In horses, the virus of glanders penetrates the body least frequently through the *digestive canal*.\* This fact has been

\* For arguments in favour of the ingestion theory of the transmission of glanders, see the remarks of Nocard (*Journal of Comparative Pathology*, Dec. 1897) and Hunting (*Veterinary Record*, 8th Jan. 1898) on that subject. McFadyean (*Journal of Comparative Pathology*, Dec. 1897) takes the same view as Friedberger and Fröhner.—TR.

proved by the generally negative results which Viborg, Gerlach, Liautard, Cadéac, Malet and others, obtained from their experiments of feeding horses on glanderous matter; although a few of these experiments gave positive results. Renault succeeded in infecting 6 horses out of 9 by feeding them on similar material. Nocard and Sadowski were repeatedly successful in transmitting glanders to horses and asses by feeding. The rare cases of the infection of sucking foals may, omitting the supposition of infection through milk, be ascribed to infection through the inspired air. On the other hand, dogs, cats, and wild animals (lions, tigers, panthers, and bears) become infected, comparatively frequently, by feeding on glandered flesh, in which cases it is possible that small injuries of the mucous membrane of the mouth may form the starting point of the invasion of the bacilli. No case of infection by the eating of glandered meat has yet been observed in man; although many glandered horses have been eaten by human beings, for instance, during the siege of Paris.

Whether cases of transmission observed in coition are those of infection of the genital mucous membrane or of infection by the inspired air, cannot always be decided. Only in a few cases have glanderous ulcers been found on the vaginal mucous membrane of mares. There is, however, no doubt that the bacilli of glanders, like those of tuberculosis, may pass from the mother to the fœtus.

Glanders may spread from a local centre in the same manner as tuberculosis. It proceeds at first by way of the nearest lymphatics. In the last stage of a chronic course, the process may be restricted for a long time to the lymph glands. By means of the blood-vessels, the bacilli may reach distant organs such as the lungs and skin. A focus of glanders in the lungs, by invading a bronchus, and by thus contaminating the expiratory air, may set up a secondary infection of the mucous membrane of the air passages of the head, larynx, and bronchi. In acute glanders, the bacilli are rapidly absorbed into the blood and thus set up a general diseased condition. We have no exact information about the part played by urine, saliva and sweat, as vehicles of the contagium.

**Varieties of Glanders.**—According as glanders is situated in the internal or external organs, we distinguish glanders of the nostrils and glanders of the lungs from glanders of the skin. In former times, the term glanders was restricted to

glanders of the nostrils and lungs; that of farcy being reserved for glanders of the skin. When the animal was affected internally and externally at the same time, he was said to be glandered and farcied.

According to its progress, we have chronic and acute glanders; the former being the ordinary, the latter the rarer kind. Acute glanders rapidly ends in death and never becomes chronic; though chronic glanders usually becomes acute. There are many causes why glanders should be sometimes acute and sometimes chronic. The chronic form assumes an acute type if the disease becomes general, that is, if the blood becomes infected with great numbers of bacilli. In the same way, ordinary chronic tuberculosis turns into acute miliary tuberculosis. As a rule, chronic glanders ends fatally in consequence of its producing a general infection of the body, that is to say, general acute glanders. Inoculated glanders, and primary glanders of the skin due to wounds, frequently manifest themselves in the acute form. The same thing occurs in those cases of glanders in which a serous infection of the wound takes place, or in which the infected animal suffers from the beginning of the attack from fever. The quantity and virulence of the absorbed bacilli and individual idiosyncrasy also seem to influence the progress of glanders.

**Anatomical Conditions in Chronic Glanders.**—The anatomical changes in chronic glanders are specific inflammatory processes which are accompanied by suppuration, ulceration, granulation, and cicatrisation. Their most frequent seats are on the respiratory mucous membrane, in the lungs, lymph glands, skin, and subcutis. Other organs are not so often invaded.

1. *The respiratory mucous membrane* is the usual seat of glanders (glanders of the nostrils). It occurs in two forms: as circumscribed nodular glanders, with the formation of ulcers and cicatrices; and as diffuse or infiltrated glanders.

*Nodular glanders*, which is the ordinary kind, is most frequently situated on the nasal mucous membrane, and is then chiefly found in the upper portion of the nasal cavity, namely, on the nasal septum, and in the cavities of the turbinate bones. The affection commences with the appearance of nodules, which vary in size from a grain of sand to a millet seed, and which are of a glassy, translucent, gelatinous condition, of a roundish or oval shape, and of a dirty-grey or greyish-red colour. These nodules project somewhat above the surface of the mucous

membrane, are surrounded by a red ring, and may attain to the size of a pea, as a maximum. Some of them are isolated, and others are arranged in groups. Microscopically they consist of a large number of lymphoid cells, which break down in the centre, with the bacilli lying between them. In consequence of the fatty and purulent disintegration, which sets in very rapidly in the centre, the nodules become yellow and change into ulcers after the purulent breaking down of their summits. These ulcers are sometimes superficial (lenticular), sometimes deep, crateriform, surrounded by a hard, prominent edge, and frequently, on becoming confluent, form large ulcers which have irregularly serrated and eroded edges and a lardaceous base. They are sometimes covered with a brownish crust. The ulcers may increase in area or in depth, and may even involve the underlying cartilage or bone, thus causing perforations of the septum nasi, distensions of the maxillary bones, exostoses on the turbinate bones, etc. The shallow lenticular ulcers may heal without leaving any visible changes; but the deeper ones, after granulating, leave a radiating, star-shaped, or ice-flower-shaped\* cicatrix, which is either smooth or horny, and which, according to the form of the ulcer, may be of an irregular oblong form. The nasal septum, or the alæ of the nostrils, are frequently covered with these scars. These ulcers and cicatrices are also found in the maxillary and frontal sinuses, in the guttural pouches, and in the Eustachian tubes, where they may produce pachydermatous thickening of the mucous membrane with accumulations of a muco-purulent secretion. They are also frequently met with in the larynx, and especially in the region of the lower vocal chords, from which the suppurating process may extend to the arytenoid cartilage and cause its partial necrosis; in the trachea, particularly on the anterior surface, on which numerous long, oval ulcers of the size of a coffee-bean up to that of a hazel nut, or long, pointed, serrated scars are frequently situated; and in the bronchi. In addition to these ulcers, a catarrhal inflammation of the mucous membrane is always present.

*Diffuse or infiltrated glanders* manifests itself as a diffuse catarrh of the mucous membrane of the nasal cavity, and neighbouring cavities, with superficial ulceration, thrombosis of the veins, inflammatory infiltration of the submucosa, considerable thickening of the mucous membrane, and the formation of a peculiar, radiating cicatrix.

\* The ice-flower is the *mesembrianthemum crystallinum*.—TR.

2. In the *lungs*, both the nodular and infiltrated forms are found.

In *nodular glanders*, we find in the lungs nodules\* varying in size from a grain of millet up to that of a pea. They are grey by transmitted light, glassy and pearl-grey by reflected light, and are surrounded by a hæmorrhagic ring. The centre of the nodules shows a pale yellow point in consequence of caseation and disintegration of the innermost cells. These nodules are of different sizes, of varying numbers, and of different ages. Along with new ones, we find caseous, calcareous (the calcification of the nodules of glanders is disputed by some), and encapsuled nodules. The formation of a capsule by a connective tissue membrane is induced by a reactive inflammation in the neighbourhood of the nodule. These nodules are either (*a*) of an embolic origin, and are then situated principally in the periphery of the lungs, the larger ones being at first red, wedge-shaped foci (infarcts), and their structure the same as that of the nodules of glanders on the nasal mucous membrane; or (*b*) they represent lobular-pneumonic foci, in which the alveoli are filled with red and white blood-corpuscles, and with desquamated epithelium of the lungs. Very soon, disintegration in the centre sets in, and, in the periphery, a reactive inflammation, which manifests itself at first by a dirty-white, lardaceous, shining, inflammatory ring, out of which a connective tissue capsule becomes developed later on. In other cases, the foci suppurate (cavities of glanders); and, finally, glanderous nodules may appear in the neighbourhood of primary foci in consequence of a glanderous inflammation of the lymphatics. Besides these nodules, we find symptoms of chronic bronchitis, peribronchitis, parabronchitis, bronchiectasis, atelectasis, gelatinous infiltration of the tissue of the lungs, and less frequently circumscribed or exudative pleuritis.

*Infiltrated glanders* of the lungs forms tumours the size of a walnut up to that of a child's head, and consists of a diffuse glanderous infiltration of the alveoli and of the interstitial connective tissue (lobar or greater lobular broncho-pneumonic foci). Frequently the infiltrated parts of the lungs very closely

\* Nocard points out that when glandered horses are treated with mallein, a certain proportion of them recover, in which case the nodules which were present in the lungs cease to contain living glanders bacilli—a fact he has fully proved by inoculation. On *post mortem* examination of the lungs, the nodules may be readily felt by passing the hand with firm pressure over the surface of the lung, which, when badly diseased, will feel like a bag full of shot or peas.—Tr.

resemble, on section, a soft sarcoma. They are of a dirty-white colour, of a gelatinous juicy consistence, and of an irregular shape. They may either become indurated, so as to form hard, connective-tissue-like new growths (fibroma-like tumours of glanders, according to Gerlach), or they may become gangrenous. In nodular glanders, and in the infiltrated glanders of the lungs, the bronchial glands, and frequently the mediastinal glands, become enlarged, indurated and studded with small foci of glanders.

3. In *glanders of the skin*, the nodules of glanders are found in the papillary layer, in the cutis, and in the subcutaneous and intermuscular tissues. The cutaneous nodules vary in size from a hemp seed up to that of a pea. They suppurate rapidly and form small ulcers. The nodules in the subcutis are inflammatory (metastatic) tumours the size of a pea up to that of a hen's egg. They change into large abscesses and burst outwards. We then find sinuous ulcers with a central cavity (crateriform ulcers), from which a purulent, lymph-like, tenacious secretion discharges. In the neighbourhood of the nodules the lymphatic vessels are inflamed, swollen, and frequently resemble a rosary or knotted cord, as they are divided by nodules of the size of a lentil to that of a hazel-nut. Ulcers often develop from these secondary nodes. The neighbouring lymph glands are at first swollen and soft; but later on they become indurated by the growth of connective tissue and studded with dirty-white nodules about as large as a pin's head, or with yellow foci of caseation. The capsule around the lymph glands becomes infiltrated with small cells, and subsequently thickened (peri-adenitis and para-adenitis). Only in rare cases do we find secondary chronic farcy, which is marked by a large diffuse new-growth of connective tissue with nodular thickening of the skin. This condition is termed glanderous elephantiasis or pachydermia. It chiefly affects the limbs and head.

4. Among the other organs, the *spleen* most frequently suffers from diseased changes in glanders. It then contains embolic nodules, which vary in size from that of a millet seed to that of a pea, or may be larger, and which usually become calcareous, or may suppurate. Similar nodules occur, though not so often, in the liver, kidneys, testicles, brain, muscles, heart, and bones. In the *bones*, a cellular infiltration of the medulla becomes developed with the formation of nodules, and purulent breaking down of the osseous tissue. Ulcers of glanders are very rare on the mucous membranes of the *eyes*, *stomach*, and *vagina*.



The *blood* shows signs of slight leucocytosis, namely, an increase of the white corpuscles. Bacilli are found in the blood only in cases of acute general infection.

**Anatomy of Acute Glanders.**—The anatomical changes in acute glanders consist chiefly in a sero-ulcerous disintegration of the respiratory mucous membrane; in a sero-sanious infiltration of the submucosa, subcutis, and intermuscular tissue; in inflammation and suppuration of the lymph vessels and lymph glands; and in metastatic formations in the skin, lungs, etc. The nasal mucous membrane, in particular, is covered with rapidly-spreading ulcers, and shows diphtheritic disintegration, and the submucosa is considerably infiltrated in the neighbourhood of the ulcers. The mucous membrane of the larynx, especially on the epiglottis and ventricles of the larynx, is phlegmonously swollen and covered with ulcers (stenosis of the larynx). Similar changes are found on the mucous membrane of the pharynx. The lungs are studded with purulent metastatic foci or fresh nodules. The skin is excessively swollen and covered with glanderous nodes. Sometimes diffuse gangrene of the skin occurs.

**Statistics.**—Röll gives the following statistics on the respective participation of the individual organs in glanders; Among 3,317 glandered horses, 1,529 (about half) manifested the disease only in the nostrils; 294 only in the lungs; and 218 only in the skin. In 846 cases, both the nostrils and the lungs were affected at the same time; in 217 cases, nostrils, lungs and skin; in 164, nostrils and skin; and in 49, lungs and skin. In 216 *post mortem* examinations at the Berlin College, the lungs were found to be free from the changes of glanders only in 10 cases; and in 173 autopsies at the Vienna College, only in 28 instances. Bollinger found the lungs to be unaffected only on 4 occasions in 52 cases of glanders which he examined *post mortem*. It appears from this that the lungs in glanders are affected in the large majority of cases.

**Symptoms of Chronic Glanders.**—The early stages of chronic glanders usually escape notice; for the disease, as a rule, runs a course of months, and sometimes of years. Owing to its insidious approach, entirely wrong views have been taken about its period of incubation, which, according to inoculation experiments, is only from 3 to 5 days.

The first symptom is a unilateral, or more rarely a bilateral *nasal discharge*, which begins in the form of small quantities of dirty white mucus (chronic nasal catarrh). This discharge may fluctuate in amount or may cease for a time. Later on

it may consist of a grey or greenish-yellow, discoloured, sticky fluid which comes from the ulcers, and of a clear, limpid or yellowish catarrhal secretion. Not unfrequently the discharge is temporarily sanious. Such hæmorrhages, which are generally slight, are often the first visible sign of old-standing cases of glanders. The source of the hæmorrhage is an ulcerous erosion of minute vessels, and the engorgement and rupture of exceedingly small veins. In a few cases, however, the bleeding is more intense, and may even cause death, especially when one of the larger pulmonary vessels inside a glandered cavity becomes corroded through and is thus opened.

*Nodules* and *ulcers* may also be found on the nasal mucous membrane, from which they may, however, be entirely absent, or may appear only at a very late stage of the disease. These nodules may also be felt in the inferior part of the nasal septum or in the conchæ, in either of which positions their stay is of very brief duration; for they rapidly change into superficial or deep-seated ulcers with eroded, banked-up, thickened borders and a lardaceous base (ulcers of the hard chancre type) from which, later on, radiating, star-shaped, or elongated scars originate.

These symptoms are associated with a *swelling of the sub-maxillary lymph glands*, which is at first somewhat diffuse, more or less hard, doughy, and slightly painful. Later on, it becomes nodular, indurated, painless, and has prominences on its surface. It is usually adherent to the lower jaw, in which case it cannot be displaced. It may also be adherent to the skin. In exceptional cases an abscess, which is generally superficial, forms in the glands. Sometimes the swelling of the glands is absent when the nasal mucous membrane is not infected, and at the beginning of the attack. It is present in almost all cases of glanderous ulceration of the nasal mucous membrane.

The *state of nutrition* becomes visibly impaired, proportionately to the duration of the disease; the patient loses condition and becomes quickly fatigued when worked; and the coat becomes dry and rough. Frequently, though by no means invariably, the animal has a cough combined with chronic dyspnoea, and appears as if broken-winded (glanders of the lungs).\* Not uncommonly the horse suffers from irregular,

\* In England we recognise the fact that grunting, without the horse being a roarer, is a symptom of chronic lung disease, which is important in the diagnosis of glanders only when other symptoms of glanders are present. This remark applies equally well to unthriftiness of coat, fever, running from the nose, etc.—TR

remittent, or intermittent fever, which is proportionate to the extension of the catarrh and ulcers, and which frequently disappears entirely when the animal is at rest. Sometimes, particularly after exercise, bleeding of the nose is set up by the ulcers eroding the walls of small blood-vessels. Internal bleeding to death and hæmaturia (glanders of the kidneys) has been observed in a few cases. Trasbot remarks on the tendency to fracture (of the ribs) which the bones of glandered horses acquire on account of their becoming brittle. Towards the end, the animal suffers from oedematous swellings of the limbs and lower portions of the abdomen and chest, and also from inflammatory swellings of the joints, testicles, and scrotum.

*Glanders of the skin* does not occur so often in chronic glanders, as in acute glanders. Its favourite seats are the limbs, shoulders, breast, and hypogastrium. The nodules or boils, which are situated partly in the skin, partly in the subcutaneous connective tissue, vary in size from a pea to a walnut, and may disappear to some extent ("fugative glanders"); although they generally change into crateriform, sinuous ulcers, the discharge from which is sticky, discoloured, and frequently sanious. Smaller superficial ulcers which are covered with a crust are more rare. The efferent lymph vessels appear to be swollen in the form of a rosary [or knotted cord], the beads of which often become ulcerated [farcy buds]. The affected lymph glands become enlarged and, later on, indurated, or they may form abscesses. It often happens that the animal becomes periodically lame from the recurrence of the affection. When, as may occur on rare occasions, glanderous elephantiasis sets in, the skin becomes enormously thickened and the underlying tissues swollen, so that the head, for instance, may assume the appearance of that of a hippopotamus. Besides this, the skin becomes studded with nodules, corrugated and extremely hard to the touch.

The progress of chronic glanders is exceedingly slow. The first visible symptom usually appears only at the end of several weeks or even months. It is even possible that glanders may be present for years in an occult state, and that the affection of the nasal mucous membrane or of the cutis may take place from the lungs by metastasis; but the anatomical changes, namely, the nodules in the lungs, may form in a few days. A distinct improvement in the disease after a long rest and with good feeding often takes place. On the other hand, an

exacerbation may occur from over-exertion, chill, etc. The duration of chronic glanders may extend to 7 years.

[The use of mallein has proved that a large proportion of glandered horses show no visible signs of the disease. Since the mallein test has been brought into use in England, this percentage has naturally become far higher than in olden times, when it was the practice to wait for ulcers to form in the nasal passages, before definitely deciding that the animal was glandered.—TR.]

**Symptoms of Acute Glanders.**—Acute glanders is comparatively rare in horses (about 10 per cent. of all the cases), although it is the usual form among donkeys and their hybrids. It may be the primary form or may develop from chronic glanders which has become generalized or complicated with acute diseases. Inoculated glanders, as a rule, assumes an acute type. Acute glanders is usually a very rapidly progressing septic infective disease, which is accompanied by gangrene of the respiratory mucous membrane and by metastatic formations in the skin, lungs, and other organs.

The affection begins with rigors and high fever, in which the temperature may rise to  $42^{\circ}$  C., and is associated with a muco-purulent nasal discharge, which, later on, becomes sero-sanious, and which is often mixed with saliva and fodder by regurgitation. The visible nasal mucous membrane is covered with nodules and ulcers which frequently become confluent, so that the entire mucous membrane finally suffers from purulent gangrene and becomes covered with diphtheritic masses. These changes on the nasal mucous membrane may take place in a very short time (two or three days). The breathing is difficult, sometimes snorting, sometimes rattling, groaning, and "roaring" (stenosis of the larynx). The following symptoms of cutaneous glanders also appear: cedematous swelling of the skin; formation of nodules and ulcers in the skin; cord-like inflammation of the lymphatics (especially in the neighbourhood of the head); and swelling and suppuration of the lymph glands, particularly those of the intermaxillary space and retropharyngeal glands. There is loss of appetite and frequently difficulty in swallowing, on account of the glanderously inflamed condition of the mucous membrane of the pharynx. Diarrhoea sets in; the urine contains large quantities of albumen; and the animal becomes rapidly emaciated and prostrate.

The progress is usually severe, and has an invariably fatal termination in from 3 to 14 days.

**Clinical Diagnosis.**—An exact diagnosis of glanders is more important and, under certain circumstances, more difficult than that of any other equine disease. The following aids to the diagnosis of glanders are usually at our command :—

1. Clinical symptoms of the disease.
2. The rhinoscope.
3. Trephining the antrum of Highmore.
4. Excision of the submaxillary glands.
5. Artificial production of fever.
6. Auto-inoculation.
7. Inoculation in other animals.
8. Cultivation of the bacilli.
9. Mallein.

(a.) Glanders may be very easily diagnosed when the chief *clinical symptoms*, which are as follows, are well marked : a purulent, tenacious and rather clear nasal discharge, especially when it is unilateral ; nodules and ulcers on the nasal mucous membrane with a lardaceous, dirty-yellow base, and raised, thickened edges ; unilateral, indurated and uneven swelling of the submaxillary glands, which are firmly adherent to the lower jaw ; farcy buds and boils on the skin, with ulcers of the skin and a corded and knotted (rosary-like) condition of the lymphatics ; phlegmonous swellings of the skin without any apparent cause, especially on individual limbs, hypogastrium, under-surface of the chest, scrotum, etc. ; rough and harsh coat ; gradual emaciation ; irregular fever ; broken wind ; cough ; periodical bleeding of the nose ; and increasing loss of strength. Frequently, however, only a few of these symptoms, or even only one is present, so that a correct decision becomes very difficult, and often impossible. A suspicion of glanders is naturally strengthened proportionately to the number of these symptoms which are present. Although a unilateral nasal discharge is highly suspicious, it is in no way characteristic. Not un rarely there is a bilateral discharge in glanders, and also a hard, elongated, painless swelling of the tracheal glands, which is also found in other diseases, chronic strangles, for instance. Even the non-suppurating condition of the swollen glands, which was formerly regarded as the great distinguishing symptom, is met with in a few rare cases of other diseases. The only exact symptom of pathogenic value in diagnosis is the presence of the characteristic ulcers with their lardaceous base, and their eroded, raised edges. It is of great practical importance to search on the mucous membrane under the alæ

of the nostrils for these ulcers, which may appear independently of any nasal discharge. We should be careful not to mistake them for accidental wounds, which are often in lines, and covered with a firm scab. The nodules and radiating, star-shaped cicatrices are also of diagnostic value. Scars resulting from injuries are irregular, usually striated and quadrangular. An irregular, remittent or intermittent fever is always suspicious, if it arises without any other apparent cause. Emaciation, unthrifty coat, broken wind, and cough are, undoubtedly, suspicious phenomena, when it is probable that infection has taken place, which fact should be borne in mind by those who own large numbers of horses.

(b.) The artificial illumination of the interior of the nostrils with the *rhinoscope* furnishes us with a valuable optical means for the exact examination of the nasal cavity, and especially of its upper portions, which could not otherwise be seen. This instrument has been used for a considerable time in veterinary practice, and has been greatly improved by Polansky and Schindelka.

(c.) *Trephining the antrum of Highmore* was first proposed in 1851 by Hertwig, and in 1859 by Haubner, as an aid to diagnosis, for which purpose it has been often used since then. It is said that this operation enables us, in cases of the disease, to find glanderous changes in the antrum. Its value, however is very much restricted by the fact that, in glanders, the mucous membrane of the antrum often remains unaffected. Besides, a non-glanderous inflammation of the mucous membrane of the antrum may produce a thickening effect on it. Also the glanderous changes, mentioned by Haubner, are frequently absent from the wound caused by the operation, even when glanders is present in the immediate neighbourhood.

(d.) The *extirpation of the submaxillary glands* (Haubner and Bollinger), with the object of examining them anatomically, is by itself of doubtful use, for the following reasons: The characteristic foci of glanders may be absent from them; simple induration of these glands also occurs in chronic nasal catarrh; in attacks of strangles, purulent foci may be disseminated in the glandular tissue; and sarcomatous and carcinomatous metastatic deposits may be found in these glands. On the other hand, it is very easy to examine bacteriologically the glanderous tubercles found in the parenchyma of the gland, so as to demonstrate the presence of the bacilli. We may consequently conclude that extirpation of these glands, combined with their bacterio-

logical examination (cultivations on potatoes), is a valuable diagnostic means with living animals (Rieck, Chelchowski, and others). According to Rudenko, the bacilli of glanders can be found in the lymph glands, even when these glands are but little, if at all, enlarged.

(e.) As far back as 1843, Bouley proposed the *artificial production of fever* as a means for hastening the manifestation of symptoms in cases of latent glanders. This procedure is founded on the long-established fact that the presence of fever tends to render the course of chronic glanders more or less acute. In recent times, Lustig recommended for this object that horses suspected of glanders should be made to work hard, and that attention should be paid to variations in their temperature, and also to the occurrence of bleeding from the nostrils. Cagny and others state that subcutaneous injections of oil of turpentine have the effect of changing chronic glanders into acute glanders.

(f.) *Auto-inoculation* (malleosation)—the inoculation of suspected animals on the abraded nasal mucous membrane with their own secretions, such as the discharge from the nostrils or from ulcers—was formerly often used to verify diagnosis. The results, however, varied greatly. In very many cases treated by Bagge, Tscherning, St. Cyr, and others, the experiments resulted in a merely local and narrowly-restricted glanderous process, or the inoculation failed entirely. It is evident that glanders causes more or less, though by no means absolute, immunity to auto-inoculation.

(g.) The *inoculation of other animals* is certainly the most valuable diagnostic means at our disposal. For this purpose, asses, horses, and guinea-pigs are the most suitable. Dogs are less so; and rabbits least of all. Löffler considers the field-mouse to be the best subject for inoculation experiments with pure cultures. Kitt gives the preference to the vole [*arvicola terrestris*] and hedgehog. The ass, if it were less expensive, would be the best of all for practical purposes; because in him glanderous inoculation is almost always followed by the acute form of the disease, which then runs a fatal course in 8 or 10 days. Next to the ass the horse is the most suitable. We must remember that negative results must not be accepted unconditionally; because the material used for inoculation may have been free from bacilli, and because the experimental subject, and especially if it be an old horse, may have been

suffering from latent glanders, and, on that account, may have failed to react. Horses, as a rule, are too costly for this purpose.

We recommend guinea-pigs on account of their cheapness and certainty of the inoculation taking effect. These animals, after virulent inoculation, manifest all the characteristic symptoms of glanders; one of the most typical in males being inflammation of the testicles. Male guinea-pigs are consequently particularly good subjects for these experiments. Their employment, however, possesses the drawback that, in their case, the average duration of the experiment will be as long as a month. Löffler recommends the following method for inoculation: The hair is removed from the lateral surface of the abdomen, and the skin is pinched up into a fold and cut with a pair of [rowelling] scissors through its entire thickness, so as to make a gaping wound of about 1 cm. in length. A previously heated steel needle is inserted into the wound, and is moved to and fro under the skin so as to form a pocket, which serves as a receptacle for the inoculation material. For this purpose we prefer to use the discharge from the glanderous ulcers, either in the form of pure pus or lumpy masses. It is well to inoculate three or four guinea-pigs for each experiment. In such cases, the first symptom is redness of the edges of the wound, and the formation of a round or oval ulcer with a suppurating, infiltrated base and a thickened edge. Towards the end of the first week the neighbouring lymph glands swell to the size of a pea, and even up to that of a chestnut, and form abscesses. Sometimes the entire process heals spontaneously from this stage. More frequently, however, inflammation of the testicles takes place during the second week, with the formation, in the testicles, of nodular spots, which develop into abscesses and discharge pus rich in bacilli. These symptoms may be associated with purulent arthritis, and cutaneous or subcutaneous nodules, which suppurate or burst. When they occur on the face, they frequently take their origin from the periosteum of the bones, and sometimes eat through, into the nasal cavity. Finally, the nasal mucous membrane, becomes affected; the animal sneezes, and breathes with difficulty; a discharge, which dries up into crusts, issues from the nostrils; and death takes place generally in the third or fourth week under circumstances of great emaciation and exhaustion. In other cases, the patient succumbs by the end of the first week, though now and then not till the eighth week. According to Cadéac and Mallet,



chronic glanders may also develop with caseous foci in the lungs, heart, liver, spleen, lymph glands, etc., and then runs a course of from 2 to 4 months. Chronic indurated glanders in guinea-pigs may sometimes heal spontaneously. Besides the described changes, we find, *post mortem*, nodules in the lungs, spleen, omentum, testicles, and retro-peritoneal tissue; ulcers in the nasal mucous membrane; perforation of the septum nasi; and partial destruction of the facial bones. The blood contains no bacilli, contrary to what is the case in the organs affected by glanders. The absence of glanders is proved just as clearly by repeated failures, with one or more animals, to communicate the disease by inoculation, as its presence is demonstrated by positive experiments (Löffler).

The **dog** has also been recommended as a suitable subject for inoculation (Pütz, Galtier, Reul, Molkentin, and others). A seton saturated with infectious secretion is placed under the skin of the nape of the neck. On the third day an ulcer, resembling a hard chancre, with a purulent base, hard edge, and irregular shape, appears on the site of the inoculation, and gradually becomes enlarged with marked symptoms of constitutional disturbance. We cannot form a positive diagnosis from these symptoms, which, in the majority of instances, are the only ones present. In rare cases, farcy buds and ulcers appear on other parts of the skin; the joints, especially those of the hind limbs, become attacked by acute inflammation, and the patient suffers from hæmorrhagic diarrhœa, purulent conjunctivitis, ulcerous keratitis, emaciation, etc. According to a personal communication by Neimann, inoculation experiments in 300 cases gave only 4 negative results. Dogs are not very suitable subjects for inoculation; because, when infected by glanders, they rarely manifest general symptoms, and in their case it is always difficult to form a correct judgment from the condition of the cutaneous ulcers.

Lisitzin and Buchner prefer the **cat** to the dog as a subject for inoculation. These animals are inoculated on the upper surface of the neck, and show from the third day a swelling at the site of the inoculation. After 5 to 7 days a typical glanderous ulcer appears in the neighbouring tissue by metastasis. The cats which died in 2 or 3 weeks showed, *post mortem*, glanders of the nose and lungs. Peuch transmitted glanders to sheep from donkeys.

**Rabbits**, though formerly much used, do not answer; because they die, after being inoculated, more frequently from septicæmia than glanders. Besides, as Friedberger has proved, a period of 2 or 3 months is required for observing them: also, the results they give are not uniform. If, after saturating the inoculation threads with nasal discharge, we draw them through the skin of the cartilage of the inside of the ear, there will form at first, purulent boils the size of a hemp seed up to that of a pea, and irregular ulcers with an uneven and suppurating base and raised edges. These ulcers sometimes perforate the cartilage of the ears. They may, however, heal spontaneously. Friedberger considers that these changes are not very characteristic. Later on, if the inoculation be successful, a discharge will issue from the nostrils, and death takes place

after about 2 months. By *post mortem* examination the lungs and spleen are found to be stuffed with glanderous nodules.

Löffler considers **field-mice** to be very good subjects for inoculation with pure cultures of the bacilli of glanders. They, however, die of septicæmia when inoculated with the nasal discharge. Death takes place in 3 or 4 days after the inoculation with pure cultures. On the first, and sometimes even on the second day, they remain apparently healthy. After that, they lose their vivacity, their hair stands on end, they cower together, refuse to eat, breathe heavily, and suddenly fall down dead without any preceding convulsions. *Post mortem*, we find on the site of the inoculation a greenish or dirty-white infiltration, from which the swollen and corded lymphatic lymph vessels proceed to the enlarged lymph glands that are infiltrated with numerous pale yellow small foci. The liver is stuffed with numbers of grey nodules, which are absent from the lungs, and which are very rich in bacilli. The nasal mucous membrane and skin are in a normal condition. The inoculation, as in guinea-pigs, is made into a small pocket on the back. Besides the field-mouse, Kitt finds the **vole** (*arvicola terrestris*) suitable for pure cultures. In this animal the spleen and inguinal glands are most frequently affected. Kitt also recommends for this purpose the **hedgehog** (*erinaceus europæus*), which becomes affected with very characteristic glanders of the spleen and lungs. Kranzfeld and Grünwald inoculated the **earless marmot** (*spermophilus guttatus*) with glanders.

(*h.*) The *bacteriological proof of the presence of the bacilli* is the most certain diagnostic means; but it can seldom be carried out in practice. Only on unopened glanderous foci (glanders of the skin, pustules of the nasal mucous membrane at the entrance of the nostrils, and in the lymph glands) can we succeed, under all necessary precautions, in obtaining a characteristic culture of bacilli on serum (minute, yellow, translucent drops), or on potatoes (a honey-coloured coating). Of all these means, the only practical one is extirpation of the submaxillary glands. Cultivations can be adopted for diagnostic purposes only in exceptional cases, as for instance in veterinary colleges (Kitt). An examination of the nasal discharge or of the secretion of an ulcer for bacilli is, as a rule, without result; because the bacilli of glanders which are in them do not possess any characteristic staining. The above-mentioned secretions swarm with numerous other bacilli which stain in exactly the same manner as the bacilli of glanders. As these secretions are also more or less decomposed, it would be very troublesome to prepare a pure cultivation from them. Consequently, the inoculation of animals, is, as a rule, the only practical diagnostic means which remains to us.

**Methods of Staining the Bacilli of Glanders.**—The technique of staining the bacilli of glanders is so unreliable, troublesome, and tedious that the practical veterinary surgeon will hardly ever undertake

it (Kitt). The following methods are recommended. 1. Löffler's first way of staining: the cover glass preparations are made in the usual manner (the material rubbed down on the glass, dried in the air, and passed 3 times through a spirit flame); placed from 5 to 10 minutes in a solution of methylene blue (30 c.c. concentrated alcoholic solution of methylene blue mixed with 100 c.c. of a  $\frac{1}{10}$ th per cent. solution of caustic potash); held for a few seconds in a 1 per cent. solution of lactic acid; rinsed in water; and examined. To make the cover glass preparation, the cover glass on which the secretion or other material is spread out is dried by being drawn slowly through a spirit flame, and is then placed for 5 minutes in the staining fluid, which consists of equal parts of aniline water, solution of gentian violet, and solution of caustic potash (1 to 10,000), after which it is dipped for one second into a 1 per cent. solution of acetic acid which has been stained a hock-yellow by the addition of a solution of tropaolin, and is immediately afterwards rinsed in distilled water. 2. Löffler has recently begun to stain the bacilli of glanders as follows: Koch-Erlich's gentian violet-aniline water solution is mixed with an equal quantity of a solution of caustic potash (1 to 10,000), or of a 0.5 per cent. solution of ammonia. The cover glass or the preparation is placed for about 5 minutes in this mixture, and is then put for not longer than 1 second in a 1 per cent. solution of acetic acid, to which as much of a watery solution of tropaolin has been added as will make the fluid assume a light straw colour, and the cover glass or the preparation is then immediately rinsed in water. 3. According to Sahlis, the cover glass, after having been duly prepared, is placed for 5 to 10 minutes in a mixture of a 1 per cent. solution of methylene blue and of a 1 per cent. solution of borax, after which it is rinsed in water or in dilute alcohol and dried.

(i.) **Mallein**, which is a preparation made from the bacilli of glanders, was first manufactured and investigated in 1891 by Kalning and Hellmann, as a means for diagnosing glanders, analogous to Koch's tuberculin. The numerous experiments which have been made with mallein during late years in Germany, Austria, France, England, Russia, Denmark, Belgium, and other countries, have not yet resulted in a conclusive decision respecting its value. Mallein can by no means claim the great practical importance of tuberculin. Contrary to the assertions of Preusse, Foth, Dieckerhoff, and others, Schütz has pointed out that mallein must not be regarded as a typical reagent for glanders.\*

In carrying out inoculation experiments with mallein (Foth's dry mallein in doses of 0.05 gramme is the safest to employ), the following points are worthy of notice. The temperature having been taken every two hours from 6 o'clock in the

\* *The Journal of Comp. Path. and Therap.*, Sep. 1898, states: "All that Schütz has proved in that connection is that some bad preparations of mallein are in use in Germany."

morning, the injection is made subcutaneously on the lower third of the right side of the neck at 10 or 11 o'clock on the same day. According to the reaction to the inoculation, the horses may be divided into the three following groups :—

1. Horses with an increase of temperature of over  $2^{\circ}$  C. and a double fever curve (Schindelka) are said to be undoubtedly glandered (?).

2. Horses with an increase of temperature from  $1.2^{\circ}$  to  $1.9^{\circ}$  C. may be suspected of glanders, and should be subjected after 2 or 3 weeks to a second or even a third inoculation.

3. Horses with an increase of temperature of not more than  $1.2^{\circ}$  C. are said to be positively free from glanders (?).

Attention must be paid to the temperature of the body at the time of the injection of the mallein; for with horses having a naturally high temperature, a much higher reaction ( $2.5^{\circ}$  to  $3^{\circ}$  C.) must be manifested before we can be certain of our diagnosis. Horses suffering from fever are said to be unsuitable for inoculation; because they either show no increase of temperature or exhibit a decrease.

The perusal of the literature on mallein teaches us that the statements regarding its diagnostic value are very contradictory. The most important point in the mallein question is the anatomical proof of the presence of glanders. As Schütz points out, it is evident that many cases of inoculation were at *post mortem* examination regarded as glanders; because there were found in the lungs, nodules which were in fact quite innocent, and which occur very often in healthy horses. Olt has proved that these nodules are mostly of parasitic origin, as they contain the lifeless embryos of echinococci. It has also been proved that other diseases react typically to mallein, as for instance, inflammation of the lungs, emphysema of the lungs, bronchitis, lymphadenitis, alveolarperiostitis, caries of the teeth, and melanosis. It has also been proved by numerous observations that many healthy horses react to mallein; and that in several cases horses which were undoubtedly suffering from glanders did not react to mallein. Finally, there is the danger, especially with bad preparations, of glanders being set up by the inoculation of mallein. Putting all this together, we must arrive at the conclusion that mallein plays at the present time only a subordinate part in the diagnosis of glanders, and that it should never be used as a diagnostic by itself, but only in conjunction with other diagnostic means.

[The weight of English veterinary experience is strongly in favour of regarding mallein as an extremely useful, though not an absolutely infallible, means of diagnosing glanders. It can be obtained from wholesale veterinary chemists either pure or diluted. The latter, which keeps good for only about a fortnight, consists of 1 volume of the former to 9 volumes of water, in which  $\frac{1}{4}$  per cent. of pure carbolic acid has been dissolved. About 20 minims of the dilute mallein is used for a hypodermic injection.

The syringe should be sterilised before and after use. The following procedure for applying the mallein may be employed: take the animal's temperature—preferably, an average of the morning and evening temperature—for the two previous days, as well as that of the day in question; inject hypodermically into the side of the neck for choice, the dose of mallein (1 c.c.); and take the temperature about 6 hours after the injection, and every subsequent 3 hours, up to, say, the 21st hour. Thus used, mallein produces under ordinary circumstances the three following reactions in infected animals—

1. A large, hot, hard and painful swelling at the seat of inoculation.
2. A well-marked rise of the internal temperature.
3. Constitutional disturbance, manifested by depression of spirits, debility, loss of appetite, "blowing," and muscular stiffness, especially of the fore extremities.

Respecting the reactionary *swelling*, McFadyean, to whom we are greatly indebted for his researches on the use of mallein, writes: "In interpreting the local reaction to mallein, attention must be paid to two points, viz., the extent of the swelling, and the period at which it reaches its maximum size. The rule as regards the first of these is that in the non-glandered horse, the swelling that forms at the seat of infection is seldom or never more than three inches in diameter, while in a glandered horse it is seldom or never less than 5 inches in diameter, and not rarely it is nearly twice that. The rule regarding the second point is that, in a non-glandered horse the local swelling attains its maximum size during the first 14 or 16 hours after the injection, and then rapidly declines, so that it has nearly or altogether disappeared by the twenty-fourth hour. In a horse suffering from glanders, the tumour continues to increase in size to about the thirtieth hour after inoculation; it persists for 2 or 3 days; then gradually recedes, and does not finally disappear until the fifth or sixth day (Nocard). Inflamed lymphatic vessels may be seen radiating from the tumour, which may be so painful as to cause lameness of the fore-leg of the side upon which it is situated." The side of the neck is chosen for the site of the injection, because it offers a flat surface for the observation of the expected swelling.

The reactionary *rise of temperature*—which under ordinary circumstances, is at least  $1.4^{\circ}$  C.; usually about  $2.2^{\circ}$  C.; and sometimes as much as  $4^{\circ}$  C.—is characterised by the fact that it remains for a considerable time at its maximum height; its principal manifestation being between the eighth and sixteenth hour after inoculation. We may consider that a horse reacts to mallein, as far as temperature is concerned, if the rise is from normal to  $39.33^{\circ}$  C.

The inutility of the mallein test in *feverish horses* (with a temperature of, say,  $38.9^{\circ}$  C. or more) is well shown in two cases reported in the *Journal of Comparative Pathology*, December 1892. The respective temperatures of these horses at the time of inoculation were  $39.5^{\circ}$  C. and  $39.66^{\circ}$  C. Their temperature after inoculation, instead of rising, fell; the condition of the swelling at the seat of inoculation was normal (being about the size of a walnut); the appetite of one of them continued good; and yet both of them were proved by *post mortem* examination to be thoroughly infected with glanders.

The reactions may occur in horses which are suffering from diseases other than glanders.

No danger of transmitting glanders is incurred by the use of the mallein employed in England; because this mallein is exposed during its preparation to a degree of heat which is amply sufficient to destroy the glanderous virus.

Injections of mallein are followed by more or less immunity to the action of this agent, consequent, apparently, on its curative influence.—  
[Tr.]

**Clinical Differential Diagnosis.**—Glanders may be mistaken for a great number of diseases, of which the following are the most important :—

1. *Simple chronic nasal catarrh* gives the most frequent cause to suspect glanders; because its course is characterised by chronic nasal discharge, swelling of the glands, and sometimes with superficial ulcers. On that account, it was formerly regarded as the first stage of glanders. A considerable time for observation is always necessary to distinguish positively between the two. Simple chronic catarrh is, as a rule, accompanied by no scars on the mucous membrane, and can be permanently cured by local treatment. Inoculation in other animals will conclusively decide the point. Cultivations on potatoes have only a doubtful value.

2. The *chronic catarrh of the accessory cavities*, especially of the maxillary sinuses, and of the guttural pouches, is frequently mistaken for glanders. Here also a long time of observation is often necessary. Besides, the curability of the affection by trephining, etc., must be taken into consideration. In cases of caries of the teeth, an examination of the teeth and of the odour (whether healthy or fœtid) from the mouth will decide the point. A case observed by Siedamgrotzky shows that caries of the teeth with chronic inflammation of the maxillary sinuses and glanders in these sinuses may be present at the same time. The presence of necrotic and tumour-like processes of the bones in the neighbourhood of the nasal cavity (ethmoid bone, etc.) may lead to a mistake. Inoculation is specially important here.

3. In *strangles* we frequently find the development of lymphangitis with formation of ulcers on the head, throat, etc. The rapid healing of the lesions and other symptoms of strangles generally render the diagnosis easy. The differentiation between glanders and chronic metastatic strangles is more difficult. Here, inoculations and pure cultivations will decide the matter. Rabe has described a case of ulcerous metastatic strangles which was very hard to diagnose, and showed ulcers in the nostrils, palate, pharynx, and guttural pouches. In-

oculations in guinea-pigs were followed by death. When the inoculated animals were bacteriologically examined, there were found in them rod-like bacteria which could not be morphologically distinguished from the bacilli of glanders. The micro-organisms were proved not to be the bacilli of glanders, only by cultivations on potatoes and by inoculation in white mice. Rabe consequently considers it absolutely necessary, after having obtained a positive result with inoculation in guinea-pigs, to make cultivations and to inoculate them in white mice in order to be certain of the diagnosis.

4. *Follicular ulceration* of the nasal mucous membrane heals rapidly and without cicatrisation. It spreads to the skin in the neighbourhood of the nostrils, and is usually complicated only with a slight swelling of the glands.

5. *Stomatitis contagiosa pustulosa* is localised chiefly on the mucous membrane of the mouth, and is distinguished by the fact of its course being benign.

6. *Injuries of the nasal mucous membrane* from external influences such as hot steam, smoke, foreign bodies, finger nails, etc., can easily be recognised by an exact examination. The subsequent cicatrices are distinguished by being generally situated on the lower end of the nasal cavity (*septum nasi*). They are oblong, striated or angular, and occur frequently in the same position on both sides. They are flat, hollowed-out, and sometimes surprisingly thick and prominent. Traumatic cicatrices have very rarely a radiating appearance.

7. *Simple bleeding of the nose*, in consequence of traumatic causes, aneurysms, angiomas, phlebitis, varicose veins, etc., of the nasal mucous membrane; hyperæmia of the head; hyperæmia of the lungs; and inflammation of the lungs, may be distinguished by an exact examination, which may have to be continued for some time. Compare also with hæmorrhage of the lungs.

8. *Carcinomata, sarcomata, actinomycosis, melanosis*, and other *new growths* of the glands of the intermaxillary space and other lymph glands. In these cases, extirpation and microscopical examination of the tumour taken in conjunction with the negative result of the potato cultivation will decide the question.

9. *Leucæmia* has sometimes a great similarity to glanders in its clinical aspect: emaciation, debility, swelling of the glands, bleeding of the nose, intermittent fever, swelling of the extremities, and of the epigastrium, etc. The differentiation is frequently possible only by an exact examination of the blood

in which a surprisingly large increase (characteristic of leucæmia) of the white corpuscles will be found. In glanders, on the contrary, the increase in the number of leucocytes is but moderate. Inoculation is valueless ; for in leucæmia no morbid secretion is available.

10. *New-growths in the nasal mucous membrane* can, in many cases, be proved only *post mortem*. Among these we may include angiomas of the septum nasi ; sarcomata and carcinomata of the nostrils and accessory cavities ; enchondromata ; rhinoscleromata ; osteomelanomata ; polypi ; and benign chronic fibrous-hypertrophies of the mucous membrane, which often closely resembles the changes of infiltrated glanders.

11. *Parasites* in the nasal cavity (*pentastoma tænioides*, and *gastrophilus nasalis*) sometimes produce the aspect of glanders in the nostrils.

12. *Lymphangitis* (simple inflammation of the lymph glands) is frequently mistaken for farcy and cannot always be easily distinguished from it. The fact that lymphangitis develops usually in the neighbourhood of wounds does not always make the diagnosis certain ; because glanders may also develop from wounds, and because the primary injuries in cases of lymphangitis may have healed up and disappeared. It is specially important to remember that lymphangitis is usually a local affection ; that farcy is a symptom of general glanders ; that lymphangitis frequently progresses very acutely with fever and great tendency to the formation of abscesses in the lymph glands ; and that farcy usually progresses slowly, without fever and with but slight participation of the glands. The abscesses which appear in the progress of lymphangitis have smooth edges and heal rapidly by granulation ; but the abscesses of glanders are crateriform non-healing ulcers. In farcy, the cords of the affected lymph vessels are narrower, as a rule, than in lymphangitis, in cases of which the neighbouring tissues generally participate in the gelatinous infiltration. To make sure of the diagnosis, an inoculation or the cultivation of the bacilli is often indispensable.

13. *Phlegmonous inflammations of the skin* on the extremities (elephantiasis), head and other parts of the body, and pyæmia, especially when it is metastatic, may give rise to a suspicion of glanders. Here also we have to depend on inoculation, because the discovery of the starting point (wounds, bruises, etc.) of the inflammation is not enough ; for glanders may also proceed from solutions of continuity.



14. *Urticaria* is fugitive, and causes neither the formation of abscesses nor the swelling of glands. The tumours of the skin which it gives rise to are benign. The same remark applies to *eczema nodosum*.

15. *Petechial fever* is distinguished from acute glanders chiefly by the slight amount of fever that is present, and by the fact that the extensive swellings of the skin are never nodular.

**Anatomical Differential Diagnosis.**—As pathological anatomy cannot be discussed in a text book of special pathology, we shall content ourselves by briefly mentioning the principal pulmonary changes for which glanders of the lungs may be most readily mistaken. We may, however, state that the macroscopical or microscopical result does not always suffice for a correct diagnosis ; but that, very frequently, the clinical and etiological conditions have also to be studied, and the bacilli sought for.

1. The lungs of old horses frequently contain, in greater or less number, calcareous or caseous encapsuled nodules the size of a lentil up to that of a pea, which contain either embolic lifeless larvæ of parasites (*echinococcus*, *sclerostomum*, etc.), and which are to be looked upon as a *phlebitis obliterans nodosa* (Olt) or as pneumonia due to very minute foreign bodies or to inhaled dust (*chalicosis s. nodosis pulmonum*). They may be distinguished from the nodules of glanders—for which they are often mistaken—by the fact that they are all about the same age ; by the absence of other glanderous lesions (especially by the absence of the enlargement of the bronchial glands), and possibly by the demonstration of particles of plants, etc., in the nodules. Csokor, Kitt, and others state that true nodules of glanders do not become calcareous. Confirmation of this statement would greatly facilitate the anatomical differential diagnosis of glanders.

2. The embolic and, as a rule, subpleural nodules (varying in size from a pea to a hen's egg) which occur in the lungs of animals suffering from strangles, pyæmia, etc., and which are sometimes scattered over the entire lungs, are mostly of the same size and age, and appear at first as wedge-shaped hæmorrhagic infarcts. As the embolic nodules of glanders possess the same characteristics ; proof of the absence of other glanderous changes or the demonstration of a non-glandered primary centre will be required to decide the point.

3. Tuberculosis of the lungs is distinguished first of all by the difference between the respective bacilli. Those of glanders, according to Csokor, are broader than tubercle bacilli and seem to be composed of small and alternately light and dark cubes. Csokor further states that the nodules of glanders consist almost entirely of lymphoid cells and that the nodules in tuberculosis are true granulation tumours which contain three kinds of cells, namely, giant cells, epithelioid cells, and lymph cells. Finally, tuberculosis of the lungs is also frequently complicated with tuberculosis of the serous membranes.

4. *Sarcomata* and *carcinomata of the lungs*, which in many cases cannot be distinguished macroscopically from the growths of glanders, can be easily recognised by aid of the microscope.

5. *Actinomycosis* and *botryomycosis of the lungs* can be mistaken only macroscopically for glanders. The microscopical appearance of the ray fungus and the blackberry-like spherical tuft of the *botryococcus ascoformans* will decide the point at once. The presence of mycelium-like threads of fungi, which may be seen by the microscope, will leave no doubt as to the existence of pneumomycosis of the lungs.

6. *Bronchial, peribronchial, and parabronchial processes*, bronchiectasis, very minute multiple foetal atelectosis, chronic interstitial-pneumonic foci, lobular catarrhal-pneumonic changes, dead echinococcus cysts, etc., may be mistaken for glanders of the lungs. The characteristic nodules of glanders and other signs of glanders are, however, absent from them.

The liver and spleen of old horses often contain multiple, calcareous, encapsuled nodules of different sizes, without other changes, which might be indicative of glanders. These nodules are emboli from the intestines, and are partly of vegetable origin.

**Therapeutics.**—A spontaneous cure in glanders, as in tuberculosis, is possible, but is of extremely rare occurrence. It is probable that reputed recoveries by the administration of medicines have no existence; although a great number of remedies have been tried, and veterinary literature, especially that of the sixties, is exceedingly rich in reports of such experiments. Compounds of chlorine, bromine, and iodine; salts of copper, mercury, silver, arsenic, and strychnine; preparations of iron, carbolic acid, alcohol, etc., have been tried. Levi and Neimann state that they have successfully used, as an intra-tracheal injection, Lugol's solution, which consists of iodine 2 parts, iodine of potassium 10 parts, and water 100 parts.

Trinchera, Uelzen, Penning and others have, however, shown that these intratracheal injections are not only useless, but that they accelerate the development of glanders. The treatment of farcy with the hot iron or with caustics seems to have been successful in a few instances (Janson). The other cases are to be looked upon very cautiously; because farcy is mostly of metastatic origin. We have to wait for further experiments on the curative effect of subcutaneous injections of creosote oil and mallein\* (Johne, Schindelka, and Semmer) before pronouncing on the value of these agents. Protective inoculation (malleosation) proposed by Bagge, Tscherning, and others, is only of historical interest.

The only rational mode of extinguishing this disease consists in the application of the severest precautionary police measures.

In some countries, glanders is regarded as an unsoundness which can be covered by special warranty. Thus, the time in Prussia, Bavaria, Württemberg, Baden, and Hesse is 14 days; in Saxony and Austria, 15 days; and in Switzerland 20 days.

[As glanders is largely imported into the United Kingdom from abroad, no horse should be allowed to be landed in this country without being examined by a veterinary surgeon, and certified to be free from this disease, except in the case of an animal brought from a glanders-free country, like Australia, and in that of race-horses, which very rarely suffer from this disease. Mr. H. G. Rogers wisely suggests the advisability, in this connection, of applying the mallein test, with a quarantine of 3 or 4 days.

With respect to the *prophylaxis* of glanders, the French method (*Journal of Comparative Pathology*, Dec. 1897, pp. 295, 296, and 345), which is as follows, is well worthy of adoption in this country, always taking into account that, under certain conditions, mallein is not infallibly diagnostic.

1. All horses presenting symptoms of apparent glanders should be tested with mallein, and if they show a reactionary rise of temperature and swelling at the site of injection, they should be destroyed. If they fail to react, they should be considered free from the disease.

2. All horses which have cohabited with glandered horses should be tested with mallein, and should be divided into two classes, namely:—  
(a) Those which give no reaction, and which consequently should be regarded as healthy as far as glanders is concerned. (b) Those which react, and which, on that account, should be rigidly segregated, and should be again tested with mallein once every month, or every 2 months. If any of this second division, after reaction, develop visible

\* Nocard (*Journal of Comparative Pathology*, Dec. 1897) has obtained several positive results with mallein.—Tr.

signs of glanders (including those of farcy), they should be promptly destroyed; and all the others which have ceased to react for 2 successive injections should be declared free from this disease.

Nocard justly points out that the manifestation of external signs of glanders is the last stage of the disease, which then has become general, and has thoroughly overcome the power of resistance of the system. Hence the treatment of such cases, even by mallein, is generally hopeless.

At present, mallein is largely employed in England for the unintentional dissemination of glanders, by owners who use it to find out the condition of their horses, and who, on obtaining the required information, sell those that react. It is almost needless to say that infected animals which manifest no outward signs of the disease, are the most potent of all means for spreading it.—Tr.]

**Glanders in Cats and Beasts of Prey.**—The cases of glanders which appear in cats, lions, tigers, etc., after feeding on glandered horse-flesh, are generally acute. The affection consists in an acute inflammation of the mucous membrane of the nasal cavity, antrum of Highmore, larynx and trachea, and is characterised by a greenish, dirty, and, later on, bloody, fetid, mucous discharge; excessive difficulty in breathing; swelling of the submaxillary glands; swelling of the nose and even of the entire head, and of the legs with lameness; formation of nodules and ulcers on the skin, with gangrenous patches of the skin the size of a mau's hand; diarrhœa; emaciation, and prostration. Death takes place in from 8 to 14 days after infection.

**Glanders in Man.**—The symptoms of glanders in man are of great importance to the veterinary surgeon. Although the predisposition to the disease is usually not very great in human beings, cases of glanders unfortunately occur now and then, especially among veterinary surgeons. Human glanders is particularly common in Russia (Woronzow and Pedkow). The parts usually affected are—the hands, nasal mucous membrane, lips, and conjunctiva. After a period of incubation of from 3 to 5 days, the infected part becomes swollen and painful, with subsequent inflammation of the lymph vessels and swelling of the glands. Fever is often the first symptom, and is almost always followed by a nasal discharge; ulcers on the nasal mucous membrane; pustules and abscesses in the skin; ulcers in the oral cavity, pharyngeal cavity, larynx, and conjunctiva; articular swellings; high fever with grave general disturbance; and sometimes intense gastro-intestinal trouble. As a rule, death takes place in from a fortnight to a month, and in some cases even in a few days. In other instances, the disease becomes chronic, with a duration of months or years. Bacteria exist in the blood in cases of acute glanders (Wassilieff). Besides the above mentioned symptoms, the correctness of our diagnosis depends on the possibility of infection having taken place, on retro-vaccination in horses or guinea-pigs, and on proof of the presence of the bacilli. Treatment is usually of no avail. The only hopeful cases are those which are purely local, and which may be cured by deep cauterisation of the glanderous centre, as we saw done in a recently infected person.

**Lymphangitis Epizootica.**—Under the respective names of epi-

zootic lymphangitis, benign farcy, African lymphangitis, and African or Neapolitan farcy, French and Italian veterinary surgeons have described a specific, infectious lymphangitis, which manifests multiple abscesses and differs from glanders. Rivolta states that this disease is caused by the *cryptococcus farciminosus*. This disease develops from wounds, after a period of incubation of about 3 months. Multiple abscesses accompanied by lymphangitis appear round the affected part in the skin and subcutis. These abscesses form nodular new growths which appear later on in the lymph glands, connective tissue of the muscles, bones, and even in the conjunctiva, and undergo a slow process of suppuration. In the skin they subsequently change into fungoid ulcers. The disease may be complicated with inflammation of the lungs, pleuro-pneumonia, pyæmia, anæmia, etc. The mortality amounts to about 10 per cent. With energetic treatment, a cure takes place in from 1 to 7 months. The affection is distinguished from glanders by the absence of the bacilli of glanders and the presence of characteristic thick ovoid bacteria which have strongly refractive outlines. The treatment consists in the excision, scraping out, or cauterisation of the purulent foci. A similar disease, which is not identical with glanders, occurs among cattle in Guadaloupe.

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#### BOVINE PLEURO-PNEUMONIA, ETC.

Bovine pleuro-pneumonia—Septic pleuro-pneumonia in calves—Infectious pleuro-pneumonia in goats—Epizootic inflammation of the lungs in American oxen.

**History.**—The first account of the appearance of pleuro-pneumonia dates from the end of the seventeenth century. It is stated that pleuro-pneumonia was observed in Hesse for the first time in 1693, and that its earliest appearance as an epizootic was at the beginning of the eighteenth century, when it prevailed chiefly in Switzerland and the neighbouring countries of Württemberg, Baden, and Alsace. We possess detailed reports on it from the year 1743. In 1751 the Sanitary College of Zürich published for owners of cattle a guide containing the chief characteristics of the disease. In 1773 the celebrated natural philosopher Haller published an essay on this disease, which was raging severely at that time, and described the symptoms, anatomical conditions, and protective measures laid down by the sanitary police. In 1735 the disease appeared in England; and in 1765 in France. From 1790 it spread over the whole of Germany, France, and Italy; and at the commencement of the nineteenth century, over all the countries

of Western Europe. During the forties it spread to America, Africa (Cape Colony), and Australia.

**Etiology.**—Pleuro-pneumonia is an infective disease peculiar to cattle. It cannot be transmitted to any other species of animal, except under certain conditions. It may be described generally as an infective inflammation of the lungs and pleuræ (*pleuro-pneumonia bovis contagiosa*). We possess no positive knowledge respecting the nature of its infective material.

The infection may be introduced into a cowshed either by diseased cattle, or, less commonly, by bearers such as cattle-dealers, attendants, utensils, fodder, dogs, etc. The sheds in busy cattle markets are very dangerous centres for the dissemination of the disease, which, for this reason, is enzootic in sugar-beetroot districts, where, on account of the great abundance of wash and beetroot parings, there is a large demand for, and traffic in, cattle. All cattle are not equally susceptible to the contagium. It is generally supposed that about 1 in 4 is immune. The disease is spread in sheds and farms principally by the respired air. Infected cattle can transmit it even during the period of incubation, before any symptoms are apparent. The disease is particularly infectious when it is at its height. The animal remains capable of transmitting the disease for 8 or 10 weeks, or even longer, after the infection, especially when necrotic foci remain in the lungs; similar to what happens in contagious pleuro-pneumonia of the horse. Walley estimates the duration of infectiousness in cases of encapsuled necrotic foci to be as long as 15 months. The disease may be conveyed by the respiratory air for as great a distance as 40 yards or more. As already stated, it is often carried by attendants, fodder, dogs, etc.; and more rarely by the flesh of slaughtered animals, which, as a rule, seems to lose its infectiousness on becoming cold. In exceptional cases, the contagium is transmitted from the pregnant cow to the foetus.

The contagium possesses considerable resistance. It may continue virulent in infected sheds for several months, and even over a year. Laguerrière proved by inoculation that portions of the lungs of cattle suffering from pleuro-pneumonia retained their infectiousness for a whole year at a temperature between 5° and 6° C. The *period of incubation* is on an average from 3 to 6 weeks; the minimum being 8 days, and the maximum 3 months. Animals which have once

passed through the disease remain immune either for several years, or for the remainder of their lives.

**Bacteriology.**—After Willems, Zürn, Hallier, Weiss, Pütz, Sussdorf, Brozzola, Bruylants, Verriest, Lustig and others had been employed in the investigation of the contagium, Pöls and Nolen in 1886 at Amsterdam found round micrococci always present in the exuded matter of the lungs. These micrococci were about  $0.9\ \mu$  in diameter, and occurred either isolated or in chains up to 6 in number. In the unstained preparations, the cocci were surrounded by a distinct envelope, which could be stained only with great difficulty, and was absent from cultivated micrococci. Glass plate cultures showed, in from 2 to 25 days at ordinary temperatures, sharply circumscribed, white, shining, and slightly prominent colonies, which had a faint yellow lustre. Stab cultures produced nail colonies which, with increasing age, assumed the colour of cream and finally became yellow. Exposure to a temperature of  $67^{\circ}\text{C}$ . for a quarter of an hour, arrested the development of these cocci. Inoculation experiments by direct injection of the cultivations into the lungs, produced, in cattle, dogs, guinea-pigs, and rabbits, an extensive pneumonia with the characteristic cocci in the exudate. Out of 100 cattle which had been inoculated in the tail with pure cultures and, subsequently, had been kept for a long time with animals suffering from pleuro-pneumonia, not one became infected.

Arloing, rejecting the theory of Pöls and Nolen, believes that he has found the exciting cause of the disease in various bacilli, especially in one which he calls *pneumo bacillus liquefaciens bovis*, which forms short, non-motile rods that, as white colonies, rapidly liquefy ("*liquefaciens*") the gelatine, and form on potatoes, at first a white, and later on a brownish layer. It occurs constantly, though only sparsely, in the fluids of the lungs along with numerous other bacteria. The bacilli, when placed in broth, emit a poisonous substance (pneumo-bacillin), which, like tuberculin and mallein in their respective actions, is stated to be a diagnostic for bovine pleuro-pneumonia; cattle suffering from this disease being much more easily affected by this agent than healthy animals. It is also stated that pure cultures render cattle immune in the same way as the ordinary inoculation for pleuro-pneumonia. Arloing believes that his experiments prove that the pneumo-bacillus is the cause of pleuro-pneumonia. Cultures from subcutaneous inoculation tumours, on being intrapulmonarily inoculated, produced the characteristic changes of pleuro-pneumonia in the lungs and on the serous membranes. As early as the fifth or sixth day, after the inoculation, deposits the size of a man's fist were found in the lungs. The diagnostic use of the lymph of pleuro-pneumonia has also been proposed by Siedamgrotzky.

**Occurrence.**—Next to rinderpest, pleuro-pneumonia is the most dangerous bovine disease. It may occur, especially at the commencement of an outbreak, sporadically; although it usually manifests itself enzootically, and not rarely epizootically. Its presence in certain districts is constant, as in Austria, France,

England,\* Belgium, and in some parts of Germany, America, Africa, Asia Minor, and Australia. The Eastern European countries, as for instance, Russia, are comparatively free, with the exception of the Western frontiers and sea-ports. The disease is most frequently met with in large herds in which there is a frequent change of cattle, and along great thoroughfares. Countries which do not import any cattle are usually free from the disease.

Pleuro-pneumonia has been observed in goats (Spinola, Koppitz, and others), camels, buffaloes, bison, and yaks. It is most likely that the reported cases of pleuro-pneumonia in pigs were founded on error. This remark also applies to the reputed cases in man, and especially in children. The fact that a form of pneumonia which occurs in children is anatomically related to pleuro-pneumonia, has no bearing on the subject. It is important to know that the flesh of animals which have died from pleuro-pneumonia can, as is the usual custom, be eaten by mankind without any injury to health.

**Statistics of Epidemics.**—1. In *Germany* during the last nine years (1886 to 1894) about 10,000 cattle (say 1,000 per annum) suffered from pleuro-pneumonia. The following figures show the great decrease in the frequency of the disease in Germany during that time: 1,778 (1886), 2,156 (1887), 1,545 (1888), 896 (1889), 528 (1890), 1,273 (1891), 1,182 (1892), 686 (1893), and 822 (1894). Of 6,024 animals which were inoculated from 1891 to 1893 in *Russia*, 34, that is,  $\frac{1}{2}$  per cent., died in consequence of the inoculation.

2. In *Prussia*, according to Felisch, 23,582 head of cattle died of, or were killed on account of, pleuro-pneumonia, during the ten years from 1876 to 1885. The figures for the respective years are as follows: In 1876, 3,117; in 1877, 1,980; in 1878, 2,098; in 1879, 2,364; in 1880, 1,749; in 1881, 1,982; in 1882, 2,079; in 1883, 3,070; in 1884, 3,252; and in 1885, 1,891. The total sum paid as indemnities within these ten years amounted to £225,000. These figures, compared with those of the last five years, show that the present laws have a restrictive effect on pleuro-pneumonia in Germany. The fact is particularly well proved by the statistics of earlier times. Thus, there occurred in Rhenish-Prussia, during the ten years from 1835 to 1844, no fewer than 100,000 cases of pleuro-pneumonia.

3. During the last 5 years there were nearly 10,000 cases of pleuro-pneumonia in *Great Britain*; † the numbers being 2,471, 2,437, 1,843,

\* For the past few years, bovine pleuro-pneumonia has been a rare disease in Great Britain.—TR.

† The following statistics show the great success which has attended the system of stamping-out as regards bovine pleuro-pneumonia in England. In the year 1890, during the last third of which compulsory slaughter was introduced, 591 cattle were found to be infected; in 1891, 778; in 1892, 134; in 1893, 30;



1,646, and 1,466, which make a total of 9,863. There is also here a steady decrease, particularly in comparison with the statistics of former times. During 1860 nearly 200,000 cattle died from it in Great Britain; and about a million during a period of 6 years.

4. In *France* the number of cases for the years 1886 to 1890 were 1,480, 1,145, 1,289, 1,700, and 2,252, showing a total of 7,866. In the department of the Nord, which is one of the chief centres of the disease, over 200,000 head of cattle died of pleuro-pneumonia in 19 years. The departments of the Seine, and Meurthe and Moselle, which is a district in the south-east, are greatly affected by this disease.

5. In *Bohemia* and *Moravia* pleuro-pneumonia is very common. In 1888 and 1889 a total of 6,600 infected cattle were reported to the authorities. According to Röhl 0.4 per cent. of all the cattle in these countries were affected from 1877 to 1887.

6. In *Hungary* 1,558 cattle were attacked during 1891 and 1892.

7. In *Belgium* there were 3,500 cases of pleuro-pneumonia from 1886 to 1890. On the other hand, the disease has not been met with for several years in *Holland*, where it was formerly constant in the Spöling district. In that centre, 600,000 cattle died of pleuro-pneumonia from 1830 to 1840.

The disease was introduced into *Australia* in 1858. During the years 1860 to 1872 one and a half millions of cattle suffered from it. At the present time it has spread over the entire continent, so that, for instance, the single colony of Queensland suffers a yearly loss of £640,000. In *America* about 10,000 cattle were attacked by the disease in the state of Illinois (Chicago) during 1886. In *Turkey* the disease appeared in the district of Adrianople in 1888. It has been reported that a disease resembling pleuro-pneumonia broke out among the ruminants of the nomad Kurds, and among buffaloes in Asia Minor.

**Anatomy.**—Pleuro-pneumonia is anatomically characterised by a progressive interstitial pneumonia with secondary hepatisation of the alveoli of the lungs, and spreading of the process to the pleuræ. Usually only one lung, the left as a rule, is infected. The anatomical changes vary according to the duration of the disease.

1. The otherwise healthy lung shows, in the initial stage, small, circumscribed, inflammatory centres the size of a hazelnut up to that of a walnut. The interlobular tissue in it is hyperæmic, permeated by single hæmorrhages and infiltrated by serum. The reddened lobules of the lungs are surrounded by bright margins, which are 1 to 2 mm. broad, and which are filled with a serous or lymphatic fluid. When the deposits are

in 1894, 15; in 1895, 1; in 1896, 9; and in 1897, 46. Past experience and the restrictions on the importation of live stock give us the assurance that this terrible scourge which formerly devastated our herds, is now only of rare and sporadic occurrence. Austria, which in 1892 adopted the system of pole-axing all pleuro-pneumonia affected cattle and all cattle which had been exposed to its contagium, is now, like England, but little troubled by the disease.—Tr.

superficial, the pleuræ become opaque and covered with slight clots.

2. At the height of the disease we find instead of the lobular inflammatory foci, a lobular pneumonia with pleuritis which is usually spread over the greater part of one lobe of the lung. The lung is considerably enlarged, of firm consistence, very heavy (weighing up to 1 cwt.), sinks in water, and does not crackle when cut into. Its section appears marbled, in consequence of the interstitial connective tissue having become thickened into broad lines, which vary in colour from orange to dirty-white, and which surround the darker-coloured lobules of the lungs. The larger lobules have a thickness of from  $\frac{1}{2}$  to 2 cm. ; and the smaller ones of from  $\frac{1}{4}$  to  $\frac{1}{2}$  cm. The colour of the enclosed lobules of the lungs depends on the duration of the process, and varies from brown-red to dirty-yellow. The recently infected lobules have a blood-red, reddish-brown, or dark brown colour (stage of red hepatisation). The colour of the older ones varies from orange to yellow (yellow hepatisation) ; and that of those of a still older date is grey (grey hepatisation). The central foci, because they are the oldest, are usually in the stage of yellow or grey hepatisation. Some of the inclosed lobules of the lungs are normal or only compressed, while others are merely hyperæmic. If we closely examine the bright interstitial lines, we shall find that they consist at first of an cedematous infiltration, which, later on, becomes plasto-fibrinous, gelatinous, and indurated, and finally tends to the formation of adventitious connective tissue. The lymph-spaces in the lines are dilated like lacunæ and filled with a serous or fibrinous fluid. In robust animals, the exudate in the alveoli is croupy and firm ; but is of a more serous character in animals of weak constitution. In the former case, a section made through the lung will be found to be granular. Besides these changes, the other lymph vessels of the lungs are dilated ; their walls are infiltrated with cells, and their lumen is in a state of thrombosis. The blood-vessels frequently show thrombi and small hæmorrhagic infarcts. The contents of the finer bronchi are often fibrinous and infiltrated with numerous white corpuscles. The bronchial glands and frequently the mediastinal glands are inflamed and swollen.

The pleuræ are covered with soft, membranous, fibrinous masses, which are sometimes lumpy or crumbling, and which can easily be detached. These deposits have a reticular surface and may attain a thickness of 2 cm. If we remove them, we

shall find the pleural vessels highly injected and suffering from well-developed ecchymoses, and the surface of the pleuræ in a rough and uneven condition. In the thoracic cavity we find, in varying quantity, a generally inodorous fluid exudate, which may be clear or opaque, and is infiltrated with flakes or lumps. Similar fibrinous masses often lie on the outer surface of the pericardium.

3. After the disease has been present for some time, we find in the affected parts of the lungs, induration, cicatrisation, caseation, calcification, necrosis, suppuration, etc. At first the interstitial infiltration becomes dense, solid and dry, and changes into firm connective tissue which makes a crunching noise \* while it is being cut with the knife. In other places we have fatty degeneration, caseation, calcification, or suppuration, in which case the enclosed lobules of the lungs, in consequence of the existing suppuration, become gangrenous, and are cast off. They then form so-called sequestra, similar to those of bones, and are surrounded by a sequestrum cavity, which has a smooth wall. The dead portions of the lungs may remain unchanged in these cavities for a long time. Frequently they become softened to the consistency of a greasy yeast-like paste. Sometimes, when they are comparatively small, they become absorbed, and the cavity shrinks and forms a scar. The hepatised lobuli of the lungs rarely regain their normal condition after the absorption of the exudate. More frequently they atrophy or collapse, undergo atelectasis, carnification, caseation, calcification or softening, become necrotic or suppurating, or form cavities. On the pleuræ we find thick and wart-like hypertrophies of connective tissue, which frequently cause the lungs to adhere to the side of the chest.

The changes in the lungs and pleuræ, which we have already described, are the most important general changes in cases of pleuro-pneumonia. It is stated that we may sometimes meet with an interstitial fibrinous exudate in the liver with atrophy of the liver-cells; sero-fibrinous effusions into the articulations, tendon-sheaths, subcutis, dewlap and brisket; intestinal catarrh; areolation of Peyer's patches; and ulcers on the gastro-intestinal mucous membrane.

**Symptoms.**—The period of incubation varies on an average from 3 to 6 weeks, with a maximum of 16 weeks and a minimum

\* Noise like that made when snow gives way under the pressure of the foot.—  
TR.

of a few days. The course of pleuro-pneumonia is usually divided into a chronic and an acute stage.

1. The period of development (chronic or occult stage) is generally characterised by symptoms of chronic lung trouble, and lasts, as a rule, about a month, although in rare cases its duration may be restricted to only a few days. During that time the lungs show merely small, lobular centres of infection. The first symptom is a short, dry, painful cough, which, at its beginning, is so slight that it is hardly audible, and occurs more often in the early morning, after the animal gets up, and is drinking or moving about, than at other times. It gradually increases in frequency and intensity, so that it becomes exceedingly distressing to the patient, which spasmodically extends its head and neck and arches its back during the paroxysms. Feeding, rumination, and the secretion of milk are partly suppressed. The animal is slightly feverish, the internal temperature varying between  $39.5^{\circ}$  and  $40^{\circ}$  C. The external temperature of the body is unequally distributed. Percussion and auscultation continue to give normal results, or we may hear in some places vesicular respiration. On the other hand, the intercostal spaces are often very sensitive to pressure. In rare cases, resolution and recovery take place at this stage.

2. The open or acute stage progresses under symptoms of high fever, acute inflammation of the lungs and pleuræ, and generally lasts 2 or 3 weeks; in rare cases, only a few days. The inflammation of the lungs spreads, and becomes lobular and diffuse. Respiration is very difficult and much accelerated. The animal breathes with widely-dilated nostrils and its flanks heave violently. It remains standing with the fore legs wide apart and the elbows turned out as much as possible, or lies down on the affected side for only a short time. The cough becomes sepulchral, and finally quite dull; and the ribs and vertebræ, especially behind the withers, become very sensitive to pressure. From the nostrils there is a mucous nasal discharge, which is sometimes mixed with blood and may become purulent and foetid. Percussion produces at first a tympanitic, later on a dull, hollow sound, which extends over a considerable portion of the lungs, and has frequently a horizontal boundary. On auscultation we hear a feeble vesicular murmur, or, if it be not present, we hear in its place bronchial respiration, ronchi, and a friction sound. In the healthy lung, the vesicular respiration is much increased. The results of percussion and auscultation coincide with the well-known results of the physical

examination which have been already discussed in connection with pneumonia and pleuritis. The temperature is from 40° to 42° C., and the rate of the pulse from 80 to 100 per minute, or more. The external temperature is very unevenly distributed. The ears and horns are sometimes warm, sometimes cold; the muzzle is dry and hot, the legs are cool, and the hair stands on end. Feeding, rumination, and the secretion of milk are entirely stopped, and thirst is increased. Usually constipation lasting several days sets in. In some cases, at the commencement of the second stage, we may observe diarrhoea with slight colicky pains, under the influence of which the animal moves about uneasily. The urine often contains large quantities of albumen. Pregnant animals not unfrequently abort. Finally, the patient becomes greatly emaciated. Œdematous swellings develop on the lower-lying regions of the body, as on the dew-lap and on the lower part of the chest and on the legs. Breathing becomes difficult, pulsation frequent and weak, and the action of the heart palpitating. The patient, not being able to stand, lies groaning with outstretched neck, and at last dies from suffocation.

**Progress and Prognosis.**—Pleuro-pneumonia is sometimes acute, sometimes chronic. In young, strong animals which have been fed on good food, it generally runs a quicker and more acute course than in old animals, or in those that have been kept on sloppy food for fattening purposes. In them the disease is slower, though more malignant. The mortality ranges from 30 to 50 per cent. Death usually takes place from 2 to 4 weeks after the commencement of the acute stage. Occasionally, in severe cases, it may occur in from 5 to 8 days. In about 30 per cent. of all the cases, recovery is incomplete, in that chronic changes, with their consequences, remain in the lungs. The total loss consequently amounts to from 50 to 70 per cent. of all the infected animals, with a minimum mortality of at least 50 per cent. Complete recovery takes place in a smaller number of cases, in which improvement commences on about the fifth day of the acute stage. Convalescence is protracted for several weeks, as the absorption of the copious interstitial exudate goes on very slowly. In a few exceptional cases the course of the disease is abortive and the symptoms mild; there being only a slight cough, trifling acceleration in the breathing, and but little loss of appetite. The changes (necrosis, carnification, adhesion to the walls of the ribs, and new growths

of connective tissue) that remain in the lungs after a typical case of the disease, cause, first of all, chronic troubles in respiration, with cough and emaciation, so that the aspect of the disease reminds one of tuberculosis of the lungs, with which malady pleuro-pneumonia is not unfrequently complicated. The disease in abortive cases has the peculiarity that relapses often occur after many months.

In large herds, at the beginning of an outbreak, only a few animals become sporadically infected ; but the number gradually increases in a few weeks. In this way the disease continues to spread in the same shed for, say, from 3 to 6 months, and may finally become enzootic in it. The intensity often varies greatly in different epizootics. In previously attacked districts the disease always assumes a comparatively mild type. In-sanitary stalls and bad food have a very unfavourable influence.

**Diagnosis and Differential Diagnosis.**—It is extremely difficult to form an exact diagnosis of the disease during its development ; because the symptoms which are present are few in number, and by no means characteristic. The slight fever and cough are the only symptoms of diagnostic importance, when the suspicion of pleuro-pneumonia has been established to some extent. In the second or acute stage, a positive diagnosis *intra vitam* can be made only when cases of pleuro-pneumonia had previously occurred, or when several occur simultaneously. As a rule, we can form a correct diagnosis only by a *post mortem* examination. The following diseases may be mistaken for pleuro-pneumonia :

1. *Non-infectious croupy inflammation of the lungs*, which may be distinguished clinically, as a rule, by its more acute and more typical course, and by its sporadic and sudden appearance ; and, anatomically, by the fact of the parts which have undergone hepatisation being all of the same age, and by the interstitial lung tissue and pleuræ being but slightly involved. Nocard states that in exceptional cases pleuro-pneumonia may run a very acute course and show hepatisation of equal ages.

2. *Tuberculosis* cannot be distinguished clinically, especially in the stage of development, from pleuro-pneumonia ; because in the former disease intermittent attacks of fever are also present in some cases. Tuberculin may be used experimentally as a diagnostic ; although it is not at all certain that animals which

have suffered for some time from pleuro-pneumonia, do not react to tuberculin. After all, an anatomical examination is the only real test. Tuberculosis and pleuro-pneumonia are frequently simultaneously present in the same animal.

3. *Traumatic pneumonia* or *pneumonia due to foreign bodies* may exhibit the same symptoms as pleuro-pneumonia. Evidence during life, of changes in the heart, caused by traumatic carditis, will be conclusive.

4. *The pectoral variety of bovine "deer and cattle disease"* may, under certain circumstances, show a great similarity to pleuro-pneumonia, on account of its epizootic appearance. The differentiation is founded on the simultaneous occurrence of the exanthematic and abdominal forms of bovine "deer and cattle disease," and on the fact that this disease, as a rule, runs a rapid course. In it, the lesions of the lungs are of the same age, and the inflammatory process is much more acute than in pleuro-pneumonia (see p. 106 *et seq.*).

5. We can rarely make a mistake in *phthisis verminalis*, *simple catarrhal pneumonia*, *pneumonia due to foreign bodies*, *pneumomycosis*, *acute emphysema of the lungs*, *atelectasis of the lungs*, *actinomycosis*, *distomatosis of the lungs*, *mercurialism*, and other diseases. The frequency of cases of foetal atelectasis is an important practical point, which has been erroneously advanced against the unrefuted fact that the essential anatomical criterion of pleuro-pneumonia is the marbled condition of the lungs, which consists in an increase of breadth of the interstitial connective tissue with hepatisation of the respiratory lung tissue of various ages, and consequently of various shades of colour. It is to be hoped that at some future period the anatomical differential diagnosis will be facilitated by the bacteriological demonstration of pathogenic micro-organisms.

**Therapeutics.**—The treatment can be only prophylactic and expectant; for the medicines which have up to the present been used, have had no curative effect in this disease. The best results are obtained from the protective veterinary police measures laid down in the Imperial [German] laws with respect to epizootics; the most important being that which orders the slaughter of all animals suffering from pleuro-pneumonia. This is the most certain means; for by it the infection is destroyed along with the infected animal. Strict disinfection with very strong agents should be carried out, and all tainted material burned. The disinfected shed should be left unoccupied as long

as practicable. Some authorities recommend that it should not be used for at least a year and a half.

Special warranty is given against pleuro-pneumonia in some countries; for instance, 40 days in Bavaria; 30 days in Saxony; and 14 days in Baden.

**Inoculation against Pleuro-pneumonia.**—Inoculation has been practised for a long time as the principal means for combatting pleuro-pneumonia. As early as the beginning of last century it was proposed in Germany by Haussmann and others. Its employment was greatly increased by the investigations which were made in 1851 by Willems in Holland, and which were published in 1852. Since that time these inoculations have been practised in nearly every country, and the literature on the subject has been very copious. In spite of all this, the question as to the value of inoculation as a protection against pleuro-pneumonia, has not yet received a final answer. The following are the chief respective arguments for and against inoculation :—

1. The advocates of inoculation, among whom we may mention Haubner, Pütz, Rueff, Bouley, Degive, Schütz, and Steffen, start from the well-known fact that one attack of pleuro-pneumonia successfully passed through, confers immunity for the remainder of the animal's life. By inoculation, a local, specific, inflammatory process, which is analogous to that in the lungs, is produced and is followed by subsequent immunity of the whole body. Inoculation also shortens the stay of the disease in a shed. The losses after caudal inoculation are stated to be insignificant. Haubner calculates that the mortality from the inoculations is from 1 to 2 per cent., and that the tips of the tails are lost in from 5 to 10 per cent. of the cases. In Holland, among 59,180 cattle inoculated in 1878 and '79, the mortality amounted to only 0.66 per cent. The experiments successfully made in Holland, Saxony, Anhalt, and Australia are cited as proofs of the benefits of inoculation. Further evidence on this point has recently been given by the positive results obtained by Schütz and Steffen. Dissemination of the affection by inoculation is denied. Inoculation has also been recommended as a far cheaper method than stamping out, which entails great pecuniary sacrifices. Degive has calculated that the results of inoculations undertaken in different countries from 1850 to 1883 show that 2.7 per cent. of 6,706 inoculated animals, and 26.9 per cent. of 2,453 non-inoculated beasts, became infected with pleuro-pneumonia,



when they were exposed to the same pathogenic conditions as regards infection. According to Pütz, the number of cases of pleuro-pneumonia was reduced in Holland, in consequence of inoculation, from 6,079 in the year 1871 to 2,227 in the year 1875; 951 in the year 1877; 157 in the year 1879; and finally to 11 in the year 1882. It is also stated that from 1850 to 1880, in Hasselt, 200,000 head of cattle were inoculated with good results, and that the losses invariably rose when inoculation was neglected. Rochebrune mentions that the Moors in Senegambia have practised inoculation from time immemorial as a preventive against pleuro-pneumonia. They perform it by inserting the point of a knife into the lungs of a slaughtered animal, and then making with the knife an incision in the skin of the nasal region:

2. The opponents of inoculation, among whom we may mention the names of Roloff, Wehenkel, Lydtin, Oemler, Zündel, Kitt, Adam and McFadyean, assert that up to the present no positive case of immunity has been proved to have been obtained from inoculation. They also point to the fact that even the advocates for inoculation are unable to give the exact duration of the immunity, and consequently make several inoculations. The specific nature of the tumour produced by inoculation is disputed; for an exactly similar tumour appears after the inoculation of pus or milk. Again, inoculation never gives rise to the form of pneumonia met with in pleuro-pneumonia, which is the chief characteristic of the disease, and becomes developed when the disease is transmitted from a pregnant cow to the foetus. Also, the results of inoculation are greatly influenced by the method and time of the inoculation, and by the quality of the inoculation material. It must frequently occur that immune animals are unintentionally inoculated, and that their immunity is credited to the effect of the inoculation. Inoculation would therefore cause the disease to be transmitted to other districts and to be artificially kept up. The opponents of inoculation state that the losses due to inoculation are often very considerable, and that the mortality sometimes exceeds that of pleuro-pneumonia. They further assert that in the documents upon which the German Imperial laws with regard to epizootics are founded, the losses due to inoculation are stated to be from 2 to 4 per cent. A French commission puts the loss of the tips of the tails at 25 per cent.; Degive gives it at from 10 to 15 per cent. To this we must add loss from decreased yield of milk, emaciation, etc., as results of the inoculation. They also point out that the disease frequently spreads in spite

of inoculation; that its progress sometimes becomes spontaneously arrested without inoculation; and that many animals pass through the disease unobserved. We must also bear in mind that in those countries in which inoculation has been most practised, the disease shows no decrease, as for instance, in England,\* where the official report lays particular stress on the doubtful value of obligatory inoculation for pleuro-pneumonia; and as in France and Belgium, in which countries inoculation is obligatory. In other countries, as in Holland, the decrease in the number of cases was not perhaps the consequence of the inoculation, but the simultaneous application of veterinary police measures, especially stamping out. Cömler, for instance, states that in Belgium pleuro-pneumonia increased in spite of inoculation from 1,481 cases in 1867 to 2,800 cases in 1878; but that it decreased rapidly in consequence of the application of strict veterinary police measures from 1,781 cases in 1880 to 1,187 cases in 1883. According to a table made by Kitt, the number of cases in England decreased only after the application of severe protective measures in 1878, during which year there were 4,590 cases. These numbers fell to 2,144 in 1879; and to 1,200 in 1882. Pleuro-pneumonia decreased in Bavaria, after the introduction of the German Imperial laws respecting epizootics, from 846 cases in 1846 to 281 cases in the year 1883. In Baden, where from 1870 to 1880 there was a yearly loss of 0.02 per cent. of all the cattle from pleuro-pneumonia, not a single case occurred in 1885.

It is difficult to draw a definite conclusion from the respective arguments of the advocates and opponents of inoculation. At any rate, inoculation seems to be useful, not as a protection, but as a means of shortening the duration of the epizootic in a large stationary herd of affected cattle. We certainly regard slaughtering the diseased animals as the best means for combatting pleuro-pneumonia, the infection of which, being unable to exist independently of the animal body, can be annihilated along with it. Stamping out, strict disinfection of the tainted sheds, and their evacuation for a long period, consequently offer a much better chance of rooting out the disease than inoculation.

**Methods of Inoculation.**—The tail is the part usually inoculated, chiefly by the following methods:—

1. *The old method.*—In order to obtain the material for the inoculation, an animal which is in the first stage of the disease is slaughtered, and the

\* See foot-note, p. 292.—TR.

affected parts are cut out of the fresh lungs. By gentle pressure exerted on the removed portions of the lungs, we obtain lymph, which we should allow to coagulate, and should then filter it through a piece of clean linen. Others use as lymph the fluid which is obtained by incisions made into the inoculation tumour on the tail, and which is said to have a comparatively mild effect. The site of the inoculation is on the dorsal aspect of the tail and about 8 to 10 cm. from the tip. After removing the hair, the inoculation is made at one or two spots with a hypodermic syringe, which should be made to penetrate the subcutis. The lymph should be clear, of the colour of yellow hock, and should never be taken from necrotic foci in the lungs, but only from those parts which are in the stage of hepatisation. The inoculation "takes," on an average, in from 75 to 90 per cent. of the inoculated animals. After a period varying from 1 to 4 weeks, there appears on the site of the inoculation an inflammatory swelling, which, under normal conditions, is not larger than a hen's egg, and which is accompanied by slight fever and accelerated breathing. If the lymph is not quite pure, extensive swelling of the whole tail with necrosis of the tip, high fever, and symptoms of septicæmia and pyæmia, will probably appear. These symptoms are also observed when the inoculation is made on the dewlap or at the root of the tail, on which account these places must be avoided for the inoculation. If the tail becomes excessively inflamed, we must prevent an unfavourable result by free incisions, antiseptic treatment, and if need be, by amputation of the necrotic tip. As already mentioned, the mortality is on an average from 1 to 3 per cent., and the loss of the tips of the tails, from 5 to 15 per cent. Sometimes a second inoculation is made after 6 or 8 weeks on a place situated above the site of the first inoculation. Martin inoculates the tail immediately under the last caudal vertebra by passing a seton 3 mm. broad and saturated with lymph. Rutherford obtained favourable results by this method, with a mortality of 2 per cent. Bad consequences are said to rarely follow this method. The swelling caused by the inoculation becomes developed between the ninth and the fourteenth day; lasts for 7 or 9 days; and disappears in from 14 to 20 days.

2. *Pasteur's method*.—A healthy calf, 2 or 3 months old, is inoculated subcutaneously with ordinary good pleuro-pneumonia lymph on the dewlap or behind the shoulder. After the animal has been killed, the lymph is sucked up by means of sterilised glass tubes of small calibre inserted into the large swelling which has become developed in the neighbourhood of the inoculated part. The collecting tubes are half filled and are then quickly closed at both ends by means of a spirit flame. Whilst the lymph is being sucked up, the free end of the tube should be closed with a stopper of wadding, to prevent the entrance of air. In these tubes, which can be sent anywhere, the lymph keeps in an attenuated form for 6 or 8 weeks. It is stated that a few drops of this lymph will suffice for the protective inoculation of a full-grown beast. By inoculating a calf from time to time, fresh lymph can always be obtained.

3. *Schütz's and Steffen's method*.—The lungs are entirely taken out of the thoracic cavity and are incised on the affected part to a depth of 1 cm. by means of a sterilised knife. The parts are torn asunder by means of the disinfected hands, and the lymph is sucked up by sterilised syringes; the site of the inoculation being on the posterior surface of the end of the tail, the hair immediately above the tuft of the tail is clipped for a width

of 10 cm., shaved off, and the part washed with soap and water, and moistened with a solution of sublimate (1 to 1000), or of creolin (1 to 20). The point of the needle is introduced through the skin obliquely inwards and downwards, and the contents of the syringe are injected into the subcutis. The punctures made by the needle are closed with cotton wool which has been dipped in sublimate collodion (1 to 1000), and the cotton wool is then fixed with a strip of adhesive plaster 50 cm. long and 1 cm. broad. The subsequent local inflammation resembles that of erysipelas. Sometimes a portion of the tail, or the whole of it, becomes necrotic and is cast off. The local reaction is more violent after the injection of warm lymph than after that of cold lymph.

4. *Intra-venous injections*.—Besides caudal inoculations, Thiernesse, Defays, Bouley, Degive, Sanderson, and others, have tried injections of 2 c.c. of the lymph into the jugular vein. The results are said to be very good and more certain than those of caudal inoculation. It is said to have given rise in one case to a marbled inflammation of the lungs.

**Septic Pleuro-Pneumonia in Calves.**—Under this name, Pöls describes an epizootic disease of calves which has a striking similarity to pleuro-pneumonia. The disease appears to be a form of septicæmia, accompanied by pleuritis and pneumonia. It runs a very acute course, with fever and great difficulty of breathing. The pathological changes in the lungs closely resembled those of bovine pleuro-pneumonia. Pöls states that the cause of the infection is a rod-like bacterium, which is very like the respective bacteria of contagious pneumonia of the pig, septicæmia of rabbits and "deer and cattle disease." Its pure cultures produce a disease very similar to contagious pneumonia when inoculated in pigs; and kill mice, rabbits, guinea-pigs, calves, and young horned cattle. We may easily distinguish Pöls' disease from pleuro-pneumonia by the microscopical demonstration of this bacterium. Galtier found the cause, in calves, of an infectious pneumo-enteritis, which occurs particularly in Southern France, to be the *pneumo-bacillus septicus*. This disease also occurs in lambs, sucking-pigs, and in a benign form in cattle, sheep, goats, and full-grown pigs, and can be transmitted by inoculation to these animals. The symptoms consist in bronchitis, pneumonia, pleuritis, inflammation of the intestines, pericarditis, and muscular degeneration. The mortality in this disease is very high. The fact of having successfully passed through one attack does not confer immunity.

Beresow has observed in Russia an epizootic pneumonia of calves with gastro-enteritis. There is lung hepatisation in foci, inflammation of the fourth stomach and intestines, ulcers in the abomasum, and swelling of the follicular mesenteric glands. Only calves of 8 weeks old and under became affected; the majority being from 3 to 5 weeks old. The disease lasts for 3 or 4 weeks. The symptoms are: cough, mucopurulent nasal discharge, suppurative conjunctivitis, diarrhœa, emaciation, and weakness.

Perroncito has described an infective pneumonia in calves, which caused great loss to farmers, and which is due, according to his researches, to the *micrococcus ambratus*. The colonies are nail-shaped, and pure cultivations on gelatine and agar-agar have an amber-yellow colour. The disease attacks calves up to the age of 3 months, and is said to occur also in sucking-pigs.

Semmer, Trinchera, Seiffert, Stöhr, Bass, Hürlimann, Imminger, Hutyra, and others, have described similar pulmonary inflammations in calves.

**Infectious Pleuro-pneumonia in Goats.**—In 1894 there appeared simultaneously in several districts in Germany an epizootic inflammation of the lungs, which had been introduced by Swiss goats from Saanen, and established itself among numerous goats in the Saxony Alps (Pusch) and in Schmalkalden (Schütz). The symptoms of the disease, which broke out soon after the arrival of the goats from Simmenthal in Switzerland, consisted in cough, dyspnœa, painful condition of the thorax on percussion, mucous nasal discharge, orange discolouration of the mucous membranes, dulness on percussion, pleural friction sound, and great weakness. *Post mortem* examination showed the presence of pneumonia and pleuritis. The disease was very infectious, and the period of incubation varied from 8 to 10 days. Some of the animals manifested a herpes-like eruption on the neighbourhood of the mouth. Pusch warns people against getting goats from Saanen in Switzerland. In Cape Colony, Hutcheon observed among goats an infective form of pleuro-pneumonia which had been introduced by Angora goats. The symptoms were those of serious chest trouble; and the disease ended fatally in two-thirds of all the cases. Protective inoculation decreased the mortality by 30 per cent. The immunity thus conferred lasted 4 to 6 months. Juquesnoz and Féris have observed similar cases. Those of Féris were supposed to have been cases of true bovine contagious pleuro-pneumonia.

**Epizootic Inflammation of the Lungs in American Oxen.**—Nocard has described an infective broncho-pneumonia among American oxen which is produced by short, motile, ovoid bacteria that are found in the hepatised tissues, in the lymph spaces of the lungs, and in the exuded matter of the bronchi. Williams considers it to be a catarrhal broncho-pneumonia which is quite independent of pleuro-pneumonia. This disease is evidently identical with cornstalk disease or cornfodder disease, which Billings and others have described, and which has been observed for many years in the Western States of America among cattle that are pastured on stubble land. Here we have a specific septicæmia which is caused by an ovoid, very motile bacterium, and which, after it has lasted for some time, becomes almost always complicated by pleuro-pneumonia. This disease was regarded in England among imported animals as pleuro-pneumonia. It can be transmitted experimentally to calves and pigs.

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#### DOURINE (*Maladie de Coût* or *Covering Disease*).

Dourine in horses—Transmission of human syphilis to animals by inoculation.

**History.**—This disease was first observed by Ammon in the Trakehnen stud in 1796. In the year 1817 it appeared in

Hanover in the stud farm of Celle and in Prussia, where it caused great losses during the thirties of last century, especially in Silesia and Poland, so that in 1840 a law containing a number of protective measures against it had to be passed. From the year 1821 we find this epizootic in Austria, especially in Bohemia and Hungary, where it was widely disseminated at times, as for instance, from 1859 to 1862 in Bohemia. In 1830 it was introduced into Switzerland and France, where it also caused very great losses among the horses of the breeding establishments; in 1836 into Italy; in 1843 into Russia; and later on into Algiers, Syria, and other countries. According to Röhl it is unknown in England and Belgium. Although it was very prevalent for some years in Germany; it has almost entirely disappeared since the introduction of the German Imperial laws with regard to epizootics. For several years not one case has been mentioned in the statistics of the German states.

Various views have been held respecting the nature of this disease. It has been commonly supposed to be identical with human syphilis, as is evident from its old names—"equine syphilis" and "chancre plague." Even in recent times it was considered to be syphilis transmitted from man to mares, which view was held in France by Bouley, Trasbot, Laguerrière, and others. The above-mentioned authors assumed that the disease was transmitted at first by syphilitic Arabs to she-asses, and from them to he-asses and mares. Laguerrière therefore proposed the name of "*syphilis des équidés*" for this disease, which is known in France as *Maladie de coït*, or *dourine*. This theory of origin is unsupported by any proof. Others state that they have seen several cases of glanders and farcy spring from dourine, and consequently they regard it as identical with glanders of the genital organs. This mistake is evidently due to the fact that in a few exceptional cases, true genital glanders was mistaken for dourine. For a considerable time, dourine was described by some authors as an independent disease of the spinal cord, the principal symptoms of which were stated to be paralysis. It was called on that account "paralytic disease" (Strauss), "nerve disease" and "breeding paralysis," which is a name that is frequently applied to it at the present day.

The fact that two entirely different diseases, namely, dourine and vesicular exanthema, have been for a long time described under the one name of "covering disease," greatly contributed to the confusion which existed respecting the nature of this malady. The old literature on the subject contains many

descriptions in which the symptoms of the two diseases are hopelessly mixed up. Only in recent times has it been found necessary to distinguish between a benign and malignant form of dourine, namely, between true dourine and vesicular exanthema, the true character of which was first recognised by Hertwig. Even at the present day we require more exact and more complete clinical, bacteriological and anatomical information concerning the etiology and symptoms of the two diseases, especially of dourine, which we shall discuss first. The investigations undertaken by von Thanhoffer deserve recognition.

**Etiology.**—Dourine is a chronic infective disease which is at first localised on the mucous membranes of the genital organs, and from thence, produces a general infection of the body. It is exclusively transmitted through infection during the act of coition from stallions to mares, and *vice versa*. Consequently, it is chiefly disseminated during the covering season, especially by stallions, and less frequently by mares. One stallion may for instance infect all the mares covered by him. The infective matter, in the form of cocci, is found, according to Thanhoffer, in the vaginal secretions, semen, spinal fluid, medullary roots of the peripheral nerves (*ischiadicus*), and in the blood. On account of the chronic course of the disease, the infective matter may remain apparently latent in the system of a horse for a very long time, even for more than a year. The disease, as Hertwig was the first to show, can be transmitted by inoculation with the secretion of the genital mucous membranes. Nocard has found that inoculations with the contents of the centres of softening in the spinal cord are successful in conveying the disease. Inoculations made by Trasbot and St. Cyr with blood, semen, prostatic fluid, and transfusions of blood gave negative results. Nocard has succeeded in producing the disease in dogs by inoculations into the anterior chamber of the eye, and found that they died after 6 to 11 weeks with symptoms of paralysis. Blaise states that he has transmitted the disease to other animals by subcutaneous injections of blood. According to Rodloff, Jessen and others, the disease is hereditary.

**Anatomy.**—The changes in the genital organs, which will be described in greater detail further on, consist in an oedematous or phlegmonous swelling of the vulva and its neighbourhood in females; and of the penis, sheath and scrotum in males. After the disease has continued for some time, the skin of the sheath

frequently becomes indurated and considerably thickened. Catarrhal swelling and thickening, ulcers, and scars may be seen on the mucous membrane of the *labia pudendi* of the vagina and of the urethra, and on the outer surface of the penis. The ulcers may be superficial or deep; and the cicatrices smooth and white, or firm and elevated. The mucous membrane of the vagina often shows circumscribed, pustular or villous hypertrophies, by which the calibre of the vagina becomes contracted. On the vulva and penis we also find unpigmented spots, corresponding to the former seats of the ulcers. The testicles are either enlarged or decreased in size and contain caseous centres of inflammation. Between the *tunica vaginalis* and *propria* we find adhesions of connective tissue and villous proliferations. The connective tissue of the epididymis and spermatic cord is often infiltrated by a yellow gelatinous exudate. The udder is sometimes inflamed, swollen, and filled with abscesses.

In the spinal cord we find, according to Thanhoffer, symptoms of myelomycosis, which manifests itself as a *myelitis hæmorrhagica centralis*, as a syringo-myelitis (a central medullary sclerosis), or as a localised degenerative process. At more or less numerous places the spinal cord is attenuated; and its transverse sections are asymmetrical, softened, and permeated with red spots. The grey matter is atrophied, and contains pulpy centres of softening of greater or less size. The spinal meninges are opaque, adhesive, hyperæmic, and have a considerable quantity of serous fluid between them. The microscopical examination of the spinal cord reveals abnormal changes in the ganglionic cells, namely, in the nucleus and body of these cells, axis cylinder processes, neuroglia (which is considerably increased), blood-vessels, etc. The membranes of the brain, especially the pia mater of the cerebrum, are hyperæmic and opaque. The ventricles of the brain contain a considerable quantity of serous fluid, and the brain substance is sometimes very œdematous. The inter-fibrillar connective tissue and the neurilemma of the larger nerves, which come from the spinal cord, are gelatinously infiltrated. The lymph glands in the neighbourhood of the genital organs are often swollen, pigmented and permeated with caseous foci the size of a pea up to that of a nut. In the abdominal cavity, the subperitoneal tissue is also gelatinously infiltrated, and the mesenteric glands may become swollen to the size of an apple. In one case Ruthe observed rounded ulcers on the intestinal mucous membrane. We find on the skin, in greater or less number and in various sizes, cir-



cumscribed, flatly-raised wheals (like those of urticaria), which are somewhat hard to the touch, and represent a serous inflammation of the cutis (vaso-neurosis). The respiratory mucous membrane undergoes a catarrhal change in some places. The cadavers are excessively emaciated and anæmic. In the lungs we sometimes find a hypostatic pneumonia or metastatic centres, if pyæmia has been present.

**Symptoms.**—The disease is characterised by local inflammation of, and by the formation of ulcers on, the mucous membrane of the genital organs, with subsequent general derangement, chiefly manifested by spinal paralysis, and vaso-neurosis of the skin (urticaria).

1. The primary local changes in the genital organs begin after a period of incubation, of, according to Maresch, from 8 days up to 2 months. The first symptom in the stallion consists in swelling of the penis, especially of the glans penis, which, later on, may become considerably swollen and paraphimosis may take place, similar to what sometimes happens after castration. On the outer surface of the penis we may see red spots, vesicles and ulcers. The *meatus urinarius* is reddened and swollen and shows a mucous discharge. The animal has a continuous desire to micturate and frequently manifests sexual excitement. The swelling also spreads from the penis to the sheath and scrotum, in which case the testicles become inflamed (*orchitis*). Finally, the inguinal glands and lymph vessels become inflamed. These local affections may, in the further progress of the disease, almost entirely disappear. In many cases nothing can be seen of the external changes just described, because the mucous membrane of the urethra is first of all affected; the only visible symptoms being strangury and, on closer examination, a mucous discharge from the urethra.

In mares the disease begins with a doughy or tense swelling of the pudenda, which swelling frequently spreads to the udder and inner surface of the thighs. The mucous membrane of the vagina is red in spots and swollen, sometimes thickened by gelatinous elevations, and covered with a turbid orange-coloured secretion. We may sometimes observe on the mucous membrane nodules, vesicles, and ulcers, which, however, are frequently absent. Also in mares the local changes may often be insignificant. The mucous membrane in the neighbourhood of the clitoris is more congested than at other parts, and the clitoris itself is swollen and erect. At the same time, affected mares

show excessive sexual excitement. They frequently suffer from strangury, and, after considerable straining, discharge urine in jets, or in small quantities, or a little sticky discoloured mucus. They incessantly shake their tails, are very ticklish, and open and close the vagina in rapid succession, while showing the clitoris in the manner common to mares in season. The discharge often assumes a corrosive ichorous condition, so that the tail and the hind legs become soiled and even scalded. In severe cases, the neighbouring lymph glands become inflamed and swollen as well as the udder, on which abscesses may appear. The swelling may even extend to the hypogastrium.

2. The general symptoms develop only after several weeks or even months ; in fact, their appearance is often delayed until the local symptoms have disappeared. At first the animals are depressed and weak ; they frequently continue to lift up their hind feet alternately, so as to try to avoid putting weight on them ; knuckle over on their fetlock joints ; lose control over the movements of their hind legs while walking ; and often drag a hind leg.

Especially stallions suffer from an attack of urticaria, in the form of sharply-defined, round, flat eminences, which may be raised the breadth of a finger above the surface, and may vary in size from a shilling up to a half-crown, or may even be larger. These eminences are caused by a serous infiltration of the papillary layer of the skin in the neighbourhood of a small artery, and are evidently of a vaso-neurotic character. They often appear and disappear rapidly and may shift their position. Usually they persist for several weeks, during which time they become moderately hard, and then slowly disappear. Their favourite sites are the croup, neck, shoulders, chest, and belly.

During the further course of the disease, a progressive paralysis of the hind quarters, combined with excessive emaciation, sets in. The animal has a staggering gait ; often gives way on the pasterns and knees ; knuckles over behind ; can raise itself from the ground only with difficulty ; and sometimes falls down unexpectedly. The affected stallion is unable to cover ; for he can neither mount a mare nor get an erection. Some patients exhibit permanent tremblings over the whole body, or local paralysis, as for instance, that of the lips, ears, and eyelids (*ptosis*). We may also observe, particularly in stallions, a striking hyperæsthesia of the skin, which manifests itself in the form of an intense pruritus, so that the animal continually rubs itself, bites the affected parts, and thus produces extensive

sores on the skin. The patient becomes extremely emaciated, especially in the hind quarters, so that the outlines of the pelvic bones and ribs become very prominent. The skin becomes dry, and the coat stands on end and loses its gloss. Some of the animals manifest pain when the lumbar region is pressed. The sensorium becomes more and more dull and blunted; and the eyes assume a staring and expressionless appearance. Towards the end the sufferer persistently maintains a recumbent position, and finally perishes from hypostatic inflammation of the lungs, septicæmia induced by the decubitus, or general cachexia. Sometimes in the final stage the patient suffers from nasal catarrh with swelling of the submaxillary glands, and conjunctivitis. Laguerrière has observed severe internal inflammation of the eyes. The appetite continues normal longer than any other function.

**Course and Prognosis.**—The course of dourine is always chronic; its average duration being from 6 months to a year, although it may extend to 4 years or even longer. In very few instances the disease runs an abortive course, in which case the morbid changes are merely local. Relapses sometimes occur, after a considerable improvement has been noticed. The local symptoms frequently disappear when paralysis sets in.

The prognosis is unfavourable, as the mortality amounts to about 70 per cent., and is more unfavourable in stallions than in mares. Recovery is possible only at the beginning of the attack.

**Differential Diagnosis.**—Before the urticaria and spinal paralysis manifest themselves, it is often very difficult to diagnose the presence of dourine on account of its chronic and exceedingly slow course. The diagnosis is more difficult in the stallion than in the mare; because the external local changes are frequently not visible in him, in which case the disease can be recognised only by the fact of the stallion infecting several mares.

Dourine is most easily mistaken for vesicular exanthema, which is an acute and usually entirely benign exanthema of the genital organs with local changes very similar to those of dourine, but without any general disturbance. Formerly, severe cases of vesicular erythema have been frequently diagnosed as dourine. The inflammation and formation of ulcers on the vaginal mucous membrane may also be mistaken for glanders, especially when the lymph vessels and lymph glands are simultaneously inflamed. In this case the further progress of the disease and inoculation

experiments will enable us to decide the point. Phlegmonous inflammation and pyæmic processes with swelling of the lymph glands, as may be sometimes observed after coition in mares and stallions which are suffering from an ichorous purulent discharge, may be mistaken for dourine. Here, also, the further progress of the disease will decide the matter. Chronic paralysis of the oins may be mistaken for the second stage of dourine.

**Therapeutics.**—Treatment can at most be successful only in the first stages of the disease, and then only by treating the local lesions with disinfectants, such as solutions, respectively, of corrosive sublimate, carbolic acid, etc. Later on, the local application of disinfectants and astringents (solutions of lead, alum, tannin, sulphate of iron, etc.) can have only a palliative effect. The external swellings of the skin, udder, and lymph glands should be treated by incisions—cantharides ointment, carbolic ointment, etc. Internally, the usual remedies for human syphilis, for instance, corrosive sublimate 0·1 to 0·2 gramme, potassium iodide 10 to 20 grammes, and arsenic  $\frac{1}{2}$  to 1 gramme, have been used not unsuccessfully, it is said. The spinal paralysis may be treated with strychnine and irritant cutaneous inunctions. It is stated that the disease has been frequently cured in stallions by castration; the supposed rationale of the operation being that, as the infection was contained in the testicles, their removal would include that of the virus.

**On the Possibility of Transmitting Human Syphilis to Animals by Inoculation.**—In spite of a great number of inoculation experiments a final answer to this question cannot be given with our present state of knowledge. It is stated that syphilis has been transmitted to monkeys, and the following positive results are claimed :—

1. In rabbits (Auzias-Turenne, Gailleton, Waller, Klebs, and others).
2. In guinea-pigs (Legros, Bradley, Michot, and others).
3. In cats (Auzias-Turenne, Bradley, and Diday).
4. In dogs (Auzias-Turenne and others).
5. In horses (Mathieu and others).
6. In pigs (Matineau, Hammonie, and others).

Against these positive results we must put a large number of negative ones obtained by Horand, and Peuch in dogs, cats, and mules; by Hunter, Ricord, Horand, Cornevin, and Teleschinski in pigs; by Wertheim, Jessen, and Tissoni in rabbits, dogs, and cats; by Hunter, Depaul, and Reynal in cattle; by Lacroix and Danet in cattle and rabbits; by Rabatel in dogs, guinea-pigs, and rabbits; by Letnick in pigs and rabbits; by Lesin in horses; and others.

Syphilis of animals has only an experimental pathological interest; because its transmission, under ordinary circumstances, from man to any

other animal has never been observed. We have never been able, especially with dogs, to accept as positively proved a case of the transmission of syphilis to one of the lower animals, in spite of various statements and suppositions. There occurs just as little in domestic animals, an infective catarrh of the mucous membrane of the urethra, namely, true gonorrhœa (clap of man); the clap of dogs being merely a purulent catarrh of the prepuce, in which the mucous membrane of the urethra does not participate.

[Metchnikoff and Roux, and Lassar of Berlin have succeeded in transmitting human syphilis, by inoculation of primary virus, to anthropoid apes. In both cases, secondaries followed the primary symptoms.—TR.]

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#### VESICULAR EXANTHEMA OF HORSES.

**Etiology.**—This vesicular eruption in horses is a generally benign and typically progressing, infective vesicular exanthema of the mucous membrane of the vagina, and skin of the vulva and penis. We place it under the present heading only because of its veterinary police relations; for it really belongs to the acute exanthemata. It is met with in many animals, most frequently in horses and cattle, and rarely in sheep, goats, and pigs. The nature of the infective matter is not precisely known. The disease can be transmitted with the greatest facility, as we ourselves have observed in our inoculation experiments. The usual mode of infection is by coition; but cases are not at all rare of mares becoming infected by other mares, without previous coition. Even foals may become infected, during sucking, on the lips, mouth, mucous membrane of the nose and eyes, and on the hind quarters (Dayot, Lundberg, Hess, and others). The infective matter is found only in the contents of the vesicles and pustules, and in the secretions of the ulcers. The disease is frequently epizootic; for one stallion may infect a large number of mares. A previous attack does not confer immunity; in fact, stallions which have recovered from the disease often become again infected after a few weeks (Steinhoff). The eruption is sometimes transmitted to man, especially to the hands of attendants, in a form which resembles small-pox, and may lead to a swelling of the entire arm, and axillary glands, and to a feverish increase of temperature. The patient, however, always makes a rapid recovery. The vesicular exanthema is consequently looked upon in France as a kind of horse-pox (Trasbot). Inoculations in cows and calves are said to produce characteristic cow-pox (Peuch and Galtier).

**Symptoms.**—The period of incubation varies from 3 to 6 days; but is often only from 12 to 24 hours. In ordinary mild cases in mares there appears, first of all, on the inner surface of the labia pudendi and on the greatly reddened mucous membrane of the vagina, dark red spots, the size of the head of a pin, which soon change into nodules, vesicles and pustules, the size of a lentil up to that of a pea. These vesicles may at first be transparent, and in their further progress may change to various shades of orange. They have a very thin covering membrane, and are found more numerous in the neighbourhood of the clitoris than on the outer surface of the swollen vulva, or on the under surface of the tail, where they also appear. The vesicles develop later on into flat ulcers which have a deeply reddened base, and which secrete a sticky, lymphatic, yellowish fluid. These ulcers often become confluent, and covered with a brown scab. On healing, they leave behind round, smooth, white cicatrices. Frequently there is present, at the same time, eczema, great sexual excitement, desire to micturate, and more or less intense vaginitis. There are no symptoms of fever or other constitutional disturbance.

In stallions the penis is swollen, externally reddened, and in some places, especially in the cervix of the penis, it is covered with pimples, vesicles and pustules, which subsequently form ulcers of greater or less depth. These ulcers on healing leave an unpigmented cicatrix. In rare cases, the eczema spreads to the outer surface of the sheath and scrotum. In one instance, Schleg observed the eruption on the inner surface of the thighs and fore-legs, and on the throat, which had probably become infected by contact with the secretion of an infected mare during coitus. There is also a discharge from the urethra; and the animals frequently stretch themselves out to stale and draw their yards. The eruption will usually heal spontaneously in mares and stallions after 3 or 4 weeks without the application of any remedies.

In severe cases the swelling of the pudenda of mares spreads anteriorly to the udder and even as far as the chest, and posteriorly to the hind legs as far as the hocks. The lymph vessels and the lymph glands become attacked and suppurate; and abscesses form in the udder, about the anus, and near the root of the tail. The ulcers become very deep, give off an ichorous discharge, and sometimes extend into the uterus. We may observe at the same time grave constitutional disturbance, high fever, stiff gait and considerable emaciation, accompanied, in

a few cases, by death (Steinhoff). The disease may last for 6 months, or even longer. A chronic vaginitis frequently remains after the inflammatory swelling has subsided. In stallions the disease may run a tedious course with swelling and abscesses of the inguinal glands, fever, digestive disturbances, and lameness.

These severe cases of the disease were formerly the frequent cause of vesicular exanthema having been mistaken for dourine, on account, perhaps, of septic or pyæmic complications having been present. The treatment in such cases is the same as for dourine (antiseptic treatment, incisions, etc.). The benign variety of vesicular exanthema may be treated by disinfectant and astringent solutions, such as those of carbolic acid, creolin, lysol, corrosive sublimate, alum, etc.

**Statistics of Epidemics.**—The statistics of the last 9 years (1886 to 1894) show that in Germany at least, the vesicular exanthema of horses is about 20 times as rare as that of cattle. Both, however, agree in the fact that they are most frequently observed in spring, namely, during the covering season. We cannot possibly prove that centres of this disease, as in other epizootics, exist in Germany. It seems, however, that during the last few years frequent cases of vesicular exanthema of cattle have been observed in the Black Forest, in Schleswig, Saxe-Weimar and Meiningen, Cassel, Wiesbaden, and Middle and Lower Franconia. During the last 9 years there occurred in Germany altogether about 1,700 cases of vesicular exanthema in horses, and 50,000 cases in cattle. The respective years show the following figures :—

Year.	Horses.	Cattle.
1886 . . .	248	3,944
1887 . . .	178	5,233
1888 . . .	200	6,874
1889 . . .	115	5,224
1890 . . .	267	5,782
1891 . . .	305	4,739
1892 . . .	145	4,969
1893 . . .	83	5,299
1894 . . .	151	8,147
	1,692	50,211

## VESICULAR EXANTHEMA OF CATTLE.

**Etiology.**—This disease, the nature of the virus of which still remains unknown, is very infectious. Frequently, all the cows of a village become infected by the same bull. Cases of transmission, independently of coition, occur more commonly than in horses. Cows often infect each other by intermediate bearers, especially when close together. For instance, the eruption has been transmitted to cows that were at the end of their pregnancy by sponges with which infected cows had been cleansed. Dinter saw the infection communicated by one cow to 9 others by the rubbing together of their hind quarters and by the whisking of their tails. Numann saw the eruption in the neighbourhood of the anus and sheath even in oxen. According to Armbruster, the infective matter is frequently transmitted to healthy cows by these animals lying with their hind quarters against infected wooden troughs. In 1885 Fenner saw the infection in 669 non-pregnant cows, and also in cows far advanced in pregnancy, and in calves of 5 weeks old. Kampmann maintains that the infection can be transmitted by the air. Schnieper, Fenner and others state that cattle may be attacked several times. One bull, for instance, was 3 times infected. Trasbot asserts that vesicular exanthema in cattle is simply transmitted horse-pox.

**Symptoms.**—The average period of incubation in cows varies from 1 to 6 days, with a maximum of 10 days. The first symptom is excessive vaginitis, the mucous membrane of the vulva and vagina is greatly congested, swollen, covered with dark red points or spots, and gives off a mucous discharge resembling the white of egg. It also develops translucent yellowish vesicles and pustules the size of a grain of millet up to that of a pea. The pustules change into ulcers, at which stage the mucous discharge becomes purulent, and dries upon the thighs and tail in the form of hard crusts. Frequently the ulcers are fairly deep and have jagged wall-like edges and a dirty base. On healing, they leave white scars, some of which are smooth; others radiating. The swollen vulva is painful to the touch, and the hind quarters are very sensitive and irritable. Considerable eczema is frequently present, so that the animals continually rub themselves and whisk their tails. They often move in a



very stiff manner, and suffer from strangury and sexual excitement.

If numerous ulcers are present, the discharge becomes very abundant, and assumes an ichorous and corrosive character, in consequence of which the neighbourhood of the vulva and the skin of the hind legs become excoriated. In a few severe cases we find necrotic lesions the size of a walnut up to that of a hen's egg, on the mucous membrane, when, for instance, an injury of the mucous membrane was present prior to the infection. As a rule, the temperature is only temporarily raised to a slight extent; but the appetite, rumination, and secretion of milk are more or less suppressed. The disease rarely causes abortion. Chronic catarrh of the vagina and permanent sterility are frequent after-consequences of this disease.

In bulls the glans penis, sheath, and scrotum are often inflamed, swollen, and painful to the touch. Pimples, vesicles, and ulcers, the size of a hemp seed up to that of a pea, form on the penis, especially on the glans, and a thin, yellowish, mucopurulent discharge flows from the urethra. Frequently strangury and phimosis are present. During erection, hæmorrhage sometimes takes place from the ulcers. The animal loses its appetite, is dull, and suffers from slight constipation and suppressed rumination. In exceptional cases extensive gangrene of the penis may take place, with permanent deformity of that organ (Kampmann).

Usually the disease lasts only for a week or two, although it may extend to a month in severe cases. The assertion lately made that the disease may assume a chronic form, lasting for several months, with granular hypertrophy of the vaginal mucous membrane, has been proved to be erroneous; for granular processes on the vaginal mucous membrane, as is well known, also occur in very many otherwise healthy cows. On the other hand, the affection may frequently continue for from 2 to 4 months in a shed. This complaint may be mistaken for foot-and-mouth disease, variola, acute and chronic vaginitis, and perhaps also for rinderpest. The treatment should be expectant. In severe cases, disinfection and astringent lotions (solutions in water of creolin, carbolic acid, alum, sulphate of copper, sulphate of iron, etc.) may be employed.

**Infectious Sexual Disease in Rabbits.**—In a superior breed of rabbits we have observed a very infectious inflammation of the external

genital organs (vulva and prepuce) which was capable of being transmitted from one sex to the other, and was distinguished by a considerable swelling of the parts and an abundant muco-purulent secretion. Treatment with a 1 to 2 per cent. solution of sulphate of copper, continued for several weeks, cured the disease.

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### ANTHRAX.

Anthrax in general—Anthrax in cattle—Anthrax in horses—Anthrax in sheep and goats—Anthrax in pigs—Anthrax in dogs and cats—Anthrax in birds—Anthrax in man.

**History.**—Anthrax is perhaps the oldest known infective disease of animals. It appears to be alluded to in the Second Book of Moses, ix. 10, as the sixth plague of Egypt. In the Third Book of Moses stress is laid on the possibility of transmitting the disease by clothes to man. The epidemic which was described by Homer in the First Book of the *Iliad*, and which raged amongst men, mules, and dogs, seems to be anthrax. Ovid has given in the Ninth Book of his *Metamorphoses* a tolerably exact description of an outbreak of anthrax. Plutarch reports that Rome was visited about 740 B.C. with a severe epizootic of anthrax. Dionysius of Halicarnassus (488 B.C.) and Livy (425 B.C.) relate examples of epizootics of anthrax in which the disease attacked, at first, cattle on pasture, then those in sheds, sacrificial animals, priests, herdsmen, country people, and finally the entire population. In Lucretius (428 B.C.) we find for the first time the name of “*ignis sacer*” applied to anthrax; and in Columella, the name of “*pustula*.” Virgil describes an infectious ovine disease which was transmitted to man in consequence of wearing the hides or wool of stricken animals, and which produced deep pustules on the skin. Pliny mentions a disease of boils in the province of Narbonne, in Gaul, at the time of the migration of nations. The Arab physicians described anthrax as “*Persian fire*.” Mezeray (966 A.D.) was the first to use the name “*Ignis St. Antonii*.” Johannes Wierus describes in the second half of the sixteenth century several epizootics in Italy (1552, 1598, and 1599), during which periods the Senate in Venice forbade the sale of beef on pain of death. Athanasius Kirchner describes in 1617 a bovine disease which infected mankind so that 60,000 people died of it. Anthrax of the tongue spread to an extraordinary extent during 1662 in

the neighbourhood of Lyons, and throughout France during 1710 and 1731. Ramazzini states that anthrax of the throat raged in 1690 in Padua amongst oxen and pigs. Anthrax appeared during 1712 in Germany (at first in the neighbourhood of Augsburg) and in Hungary; during 1726, in Poland, Silesia, and Saxony; during 1731 and 1757, in France as gloss anthrax, or carbuncle of the tongue, among almost all the domestic animals (horses, donkeys, cattle, sheep, pigs, deer, dogs, fowl, fish) and men. In 1755 and 1761 it appeared in Franconia; in 1758 and 1759 in Finland and Russia; in 1774 in Guadeloupe (West Indies). Chabert demonstrated in 1780 that the different kinds of anthrax were really one and the same disease. He classified and named them in a manner which has been followed up to the present day. Kausch published in 1805 a good description of anthrax, but denied its contagiousness. Since then, we may mention, in particular, the invasions of anthrax in the years 1807, 1810, 1819, and 1827, from which time a gradual decrease of the disease has taken place. Delafond and Gerlach (1845) investigated ovine anthrax in a very careful manner. Although the contagiousness of the disease was not recognised by Delafond, it was experimentally proved by Gerlach. Heusinger published in 1850 a comprehensive treatise on anthrax, especially from an historical and geographical point of view. He regarded anthrax as a malaria neurosa.

The knowledge of anthrax was greatly advanced during the fifties of last century. In 1855, Pollender, of Wipperfürth, published the discovery, which he had made as early as 1849, that an innumerable number of extremely fine rods existed in the blood of anthrax-stricken cattle. These rods were independently observed in 1850 by Davaine (Paris), and in 1857 by Brauell (Dorpat). Brauell found the rods in the blood during life, and used them as a diagnostic and prognostic factor; but he denied that they represented the virus of anthrax. It was only in 1863 that Davaine declared that they were the cause of anthrax, and were bacteria, against which theory Bouley, Sanson, and other authorities, at once protested. Cohn was the first to call these rods bacilli, and to suspect the existence of permanent spores. To R. Koch is due the credit of having demonstrated the development of these permanent spores from the rods and the transformation of the spores to bacilli. He also made pure cultivations of the bacilli, and clearly explained the biology of the bacillus anthracis. Protective inoculation against anthrax, which was introduced by Toussaint, and was widely advanced

by Pasteur and others, is also of great scientific and practical interest.

**Bacteriology.**—Anthrax is an infective disease that is caused by a bacterium, the bacillus anthracis, which belongs to the rod-like schizomycetes, and is specially characterised by producing resisting spores.

1. The *bacilli of anthrax* present themselves under the microscope in various forms, according to the methods used in preparing and staining the preparations. In fresh, unstained drops of blood they form limpid, non-motile rods with rounded ends, and may then be easily mistaken for other bacilli (the bacilli of putrefaction). In stained cover glass preparations we see the bacilli from 1.5 to 2  $\mu$  long, and from 1 to 1.5  $\mu$  broad, attached to one another, mostly in the form of a chain, which is from 5 to 20  $\mu$  long. The straight, slightly bent, or kinked chain-rods show a very characteristic method of division; as there are present at the place of union of the individual bacilli, transverse, fine, colourless luminæ by which the individual links seem to be transversely cut off. The ends of the individual bacilli in the chains assume various forms according to the method of staining. They appear to be connected sometimes in a straight line, and at other times in a bi-convex or bi-concave series, like the segments of a bamboo cane. Lately, special importance has been ascribed to the presence of an area which can be but very little, if at all, stained, or of a capsule-like covering, or membrane, in the neighbourhood of the cell-body of the individual bacilli. The bacilli of anthrax are to be found in all parts of the body, especially in the internal organs. They increase in the living body by transverse division and longitudinal growth (asporogenous bacilli).

Outside the animal body the bacilli form long filaments, which are often twisted and intertwined in various ways. They never ramify and may become 100 times as long as the original rod. The contents of the threads gradually become finely granulated, and there appear in it in regular order highly refractive and sharply defined oval bodies, namely, the spores, which become free after the disintegration of the threads (sporogenous bacilli).

The bacilli of anthrax can be cultivated on different nutrient materials, such as upon and in gelatine, in fluid nutrient substrata, on potatoes, agar-agar, etc. Surface cultures on gelatine form dirty-white, cloudy opacities, which liquefy gelatine.

When we use a weak magnifying power, we find the cultivation to be composed of a confused mass of threads, which pass over the edge of the colony and return, and resemble whipcord. Stab cultures in gelatine show, in the track of the needle, a dirty-white line, which gives off radiating branches or thin sprouts, and consists apparently of a fine crystalline structure (bacilli); and the gelatine liquefies. Potato cultivations produce a pale, dirty-white covering, in which the rods bear oval spores. On agar-agar they form white, bluish, and slightly-shining cultivations; and in fluid nutrient substrata (blood serum), grey, flocculent layers which lie on the bottom of the tube.

The bacilli are stained very easily with basic aniline colours, especially with an alkaline solution of methylene blue; but the spores do not take this stain. If we wish, on the other hand, to stain only the spores, the preparation must be previously treated for 15 seconds with concentrated sulphuric acid and then carefully washed. The bacilli stain by Gram's method.

**The Biological Relations of the Bacilli of Anthrax** are not less important than the morphological relations already described. Here we have to specially consider the influence of certain nutrient materials, of oxygen, of temperature, and of certain antiseptic and disinfectant substances.

(a) The following *nutrient mediums* may be mentioned: blood, blood plasma, blood serum, transuded matters of the body, aqueous humour, milk, and other mammalian secretions and excretions, animal substances and excretions which are mixed in the soil, especially alkaline fæces of cattle, neutral or slightly alkaline meat broth, meat peptone gelatine, raw and boiled potatoes, slightly alkaline infusions of hay, infusions of many grasses, with the addition of prepared chalk, which neutralises their acids, infusions of pea-straw, juice of carrots, turnips, swedes, beetroot and potatoes, and crushed cereals, particularly wheat and legumes. A certain amount of water, which must be neither too large nor too small, is necessary for the development of the bacilli. A larger proportion of water than 1 to 20 hinders their development. Thus the bacilli die in a day in distilled water and in cool spring water (Hochstetter). They perish in the ordinary temperature of a room or of an incubator (Meade Bolton), in water which is considerably mixed with organic matter, and at the temperature of well water (Wolffhügel and Riedel). The bacilli, in thin layers of tissue and blood, are destroyed by drying in 12 to 30 hours; in moderately thick layers, in 2 or 3 weeks; and in very thick layers, in 4 or 5 weeks.

(b.) The *oxygen* of the air is indispensably necessary for the development of the bacilli of anthrax, as they are aerobic. For this reason, spores never develop in the interior of an anthrax cadaver which does not contain sufficient oxygen for that purpose (Koch). Johnes states that inside the flesh of a skinned and cut up animal, neither filaments nor spores develop, even if the flesh is exposed for several days to a rather high summer temperature. For the same reason, anthrax blood enclosed in

tubes loses its power of infection after 8 days. On the other hand, spores and bacilli form on the surface of the flesh of animals which have died of anthrax, and which have been cut up into joints, if from the beginning there has been a sufficiency of moisture and warmth. Boschetti found very virulent bacilli after 8 months in meat prepared in this manner, and Savarese found them in a sausage which had been made of anthrax flesh 4 months previously. According to Semmer, spores also form in the internal organs.

(c) Temperature is a very important condition in the development of the bacilli, the most favourable being one of  $35^{\circ}\text{C}$ . Temperatures above and below that standard either retard the action or nullify the virulence of the anthrax bacilli, the development of which ceases entirely at  $12^{\circ}\text{C}$ ., and also if the bacilli are buried deeply in the ground; because the temperature of the ground is always under  $12^{\circ}\text{C}$ . Development is completely arrested at  $45^{\circ}\text{C}$ ., and death takes place under the prolonged influence of a temperature of  $55^{\circ}\text{C}$ . For the preparation of his inoculation material, Pasteur has taken advantage of the fact that exposure to high degrees of temperature decreases the virulence of these microbes. With this object he cultivated for a long time the bacilli under oxygen at  $42^{\circ}$  to  $43^{\circ}\text{C}$ . Toussaint produces his inoculation material by warming the bacilli up to a temperature of from  $50^{\circ}$  to  $55^{\circ}\text{C}$ . The bacilli are killed by cold only in very rare cases. According to Feser, the cold of winter does not affect their vitality unless it falls under  $-10^{\circ}\text{C}$ . and continues as low as that for at least 3 days. Gibier prepared attenuated inoculation material by cooling the bacilli down to  $-45^{\circ}$ . Klepzw states that a three days' exposure to a temperature of  $-26^{\circ}\text{C}$ . does not destroy the bacilli.

(d) Arloing states that *light*, especially sunlight, impedes the growth of the bacillus.

(e) The bacillus of anthrax is destroyed by *putrefaction*, if exposed to its influence for a long time. Feeding experiments have shown that it is killed by normal gastric juice. Even spore-containing bacilli are killed by gastric juice in half an hour (Straus and Wurtz).

(f) *Chemical agents* influence the bacilli in various ways, either by checking their development or by killing them. Thus, development is stopped by corrosive sublimate (1 to 300,000—1,000,000), mustard oil (1 to 33,000), potassium arsenicate (1 to 10,000), iodine (1 to 5,000), bromine (1 to 1,500), salicylic acid (1 to 1,500), carbolic acid (1 to 1,000), boric acid (1 to 800), quinine (1 to 600), etc. The bacilli are killed by corrosive sublimate (1 to 30,000), formaldehyd (1 to 20,000), creolin (1 to 15,000), carbolic acid (1 to 100–200), thymol and salicylic acid (3 to 1,000), permanganate of potash (1 to 1,000), sulphurous acid, alcohol, etc. On the other hand, the bacilli appear to be unaffected by either the external or internal employment of iodoform. Forster states that the bacilli die in from 18 to 24 hours if common salt is sprinkled on them, and in less than 18 hours when the spleen, blood, etc., of animals affected with anthrax are salted. He also says that the spores retain their vitality after having been salted for months. The same remark applies to brine for pickling meat (Abel).

2. The *spores of anthrax* are formed only outside the animal body, by the free ends of the bacilli becoming continually elon-

gated. The protoplasm of the filaments which have by this time sprung up, becomes granulated and the spores make their appearance for the first time in the form of very minute and highly refractive granules, which gradually enlarge into true oval spores. The bacilli are produced from the spores by the spores becoming elongated in the direction of their long axis towards one side in the form of an oval, cylindrical process. This process gradually grows into a bacillus, and the spore becomes disintegrated and finally disappears. If from an infected animal the spores get into the soil, they may remain there in a state of arrested development for many years; ten or even more. The spores, quite independently of the animal body, may however vegetate in the soil and surface water, which fact is of great practical importance; for it explains the manner in which the disease becomes enzootic in certain districts, in which the contagium may retain for many years its capability of becoming developed without the importation of fresh cases being necessary for its appearance. We also see how the disease may spread in any direction by the instrumentality of feeding materials, soil, flowing water, inundations, etc. The anthrax bacillus is consequently not only an endogenous, but is also an ectogenous bacterium. Under the latter condition it produces a miasmatic; and under the former, a contagio-miasmatic infective disease.

[Observation of the enzootic manner in which anthrax occurs in certain tropical and semi-tropical parts of the world, such as Eastern Bengal, Manipur, and Burma, in certain years and seasons, among different kinds of animals, as for instance, horses, cattle, and buffaloes, apparently without the agency of infection of one animal to another, strongly supports the theory that, under favourable conditions of soil and climate, anthrax bacilli can maintain a vigorous ectogenous existence. In countries like England, on the contrary, their life outside the animal body is probably that of exotics.—TR.]

**The biological relations of the spores of anthrax** differ from those of the bacilli. Oxygen and a certain degree of heat are also necessary for the formation of spores; the most favourable temperature being 35° C. The temperature limits are 12° and 43° C. An abundant formation of spores takes place as early as 2 days with a temperature varying from 20° to 25° C., and consequently under the influence of summer heat, or in the ordinary temperature of a dwelling room. According to Arloing, light (especially that of the sun in summer, and even in November, according to Ward) impedes and even entirely arrests the development of both bacilli

and spores. The spores offer much more resistance to various injurious influences than the bacilli. Thus they remain alive in water for an exceedingly long time, and for many years when in a dried condition. They can successfully bear exposure to  $110^{\circ}$  C. for 10 minutes or to  $-110^{\circ}$  C. for several hours. Putrefaction takes a long time to kill them. The best disinfecting agents for destroying the spores are corrosive sublimate, creolin, chlorine, bromine, and iodine. They are killed by a 1 to 1,000 solution of corrosive sublimate in 10 minutes; by a 2 per cent. watery solution of chlorine, bromine, or iodine in 1 day; and by a 3 per cent. solution of creolin in 48 hours (Eisenberg). It is also stated that formaldehyd (1 to 1,000) kills them in an hour. The spores remain virulent after having been placed for 20 days in a 5 per cent. solution of creosote; for 19 days in a 10 per cent. solution of lysol; or for 12 days in a 5 per cent. solution of carbolic acid (Lignières).

**General Pathogenesis of Anthrax.**—It is very improbable that the bacilli and spores of anthrax ever pass directly from one animal to another. Anthrax is not a directly infectious disease; although the virus may adhere to any part of the body, especially by means of secretions and excretions. When the transmission of anthrax from one animal to another occurs, it is almost always effected by intermediate bearers, such as utensils, people, and insects. The great majority of cases of anthrax is caused by a miasmatic infection from the soil. Here we must recognise a three-fold mode of immigration of the infective matter, namely, by the alimentary canal, by the skin and the natural openings of the body, and by the lungs. In cattle, the bacteria are usually received by the alimentary canal; and in horses and sheep by the skin, or by the intestines.

1. *Infection of the intestines*, which is the usual form, comprehends those cases of anthrax to which the names of "intestinal anthrax," "fodder anthrax," "spontaneous anthrax," "internal anthrax," "anthrax without external manifestations," "anthrax without localisation," "anthrax fever," etc., have been given. The spores, and in less number the bacilli, are received by the animal from the food and drinking water; the chief port of infection being the small intestine, the mucous membrane of which need not necessarily be injured. Although the acid gastric juice, as in tuberculosis, kills most of the bacilli, it is unable to injuriously affect the free spores. The principal vehicles for the spores in intestinal anthrax are those feeding materials which have been grown on or near places in which animals suffering from anthrax have died, or have been superficially buried, or have deposited their excrements; and fodder obtained from infected districts. Some of the spores which are



contained in the surface of the soil may become, by rain or dust, attached to various food-plants, or some of the infected soil may adhere to them, as for instance, to potatoes, turnips, etc. Water which carries the spores into the intestinal canal may, like fodder, be taken from infected places, such as wells, swamps, bogs, ponds, puddles, etc., which were contaminated by dead bodies of infected animals, tanneries, wool-washing establishments, etc. The infection may also be imported in artificial manures, unburnt bone powder, hair manure from tanneries, blood manure, etc. Carnivora may become infected by feeding on the dead bodies of diseased animals; and sucking animals, by infected milk. The spores may be introduced by the renewal of the flooring of the stall or of the litter by, respectively, infected earth or infected bedding. Pasteur's supposition that the spores were brought by earth-worms, which, according to Darwin, bring up soil to the surface from below, and thus cause a constant renewal and mixing of the superficial crust of the earth, was declared by Koch to be untenable; because, as he said, the temperature of the ground at a depth of from  $\frac{1}{2}$  to 1 metre was too low for the formation of spores, and because it was experimentally demonstrated that the contents of the intestines of worms which had been kept in anthrax soil were proved by inoculation to be non-poisonous. Bollinger has, however, experimentally proved the correctness of Pasteur's view. Five per cent. of the earth-worms of an anthrax pasture sent to him was found to contain the contagium of anthrax. As Karlinski, Proust, and others have shown, the spores of anthrax may also be disseminated by slugs and even by insects and larvæ which are found on untanned, infected skins (*dermestes vulpinus*), and which excrete anthrax spores in their fæces.

2. *Infection of the skin* is the cause of those cases of anthrax to the local manifestations of which, "carbuncle disease" and other names have been given. This mode of infection is comparatively rare and appears only sporadically. In such cases, the bacilli and spores penetrate through wounds in the skin and mucous membranes of the openings of the body, while the animals are grazing, and by means of infected utensils, bites of shepherds' dogs, blood-letting, infected instruments in operations, skins (even tanned skins), insects, especially the *musca domestica* and *vomitoria*, and various kinds of gad-flies and simuliæ. Bollinger and Zeilinger succeeded in producing anthrax in rabbits by inoculating them with flies which

had been caught on an anthrax cadaver. Anthrax has been experimentally produced by rubbing cultivations of anthrax into the skin of guinea-pigs (Machnoff).

3. *Inhalation-anthrax* is the rarest form. In it the spores penetrate through the lungs into the body. It has been experimentally proved that healthy organs of breathing can be infected in this manner (Feser, Buchner, Lemke, and Enderlen).

Besides these 3 kinds of infection, it seems possible that the disease may be transmitted from the mother to the foetus (placental infection). Latis, for instance, succeeded 8 times out of 15 cases in transmitting anthrax to the foetus through the mother in guinea-pigs. Malvos and Rosenblath believe that their inoculation experiments prove the transmissibility of the bacilli of anthrax from the mother to the foetus. Although the placenta is normally impermeable to bodily elements, it becomes patent under the injurious influence of the anthrax bacilli, which, if we may use the expression, grow through from the maternal side into the foetal side of the placenta (Birch-Hirschfeld).

The dissemination of the spores and bacilli in the body takes place in anthrax of the skin, first of all, by local increase of the bacilli in the skin, and subcutaneous connective tissue. The dissemination in the firm cutis is slow; the bacilli migrating, according to Siedamgrotzky, in the skin at the daily rate of only about 5 mm. Consequently, from a cutaneous infection of inoculation, only a carbuncle arises; although from a subcutaneous or submucous infection the bacilli can spread rapidly and produce the so-called oedema of anthrax. The further dissemination in the body takes place chiefly by the lymphatics and blood-vessels. It is somewhat peculiar that the anthrax bacilli can be found in the blood only a very short time before death. It seems when the anthrax bacilli first penetrate from the local infected part into the blood, they fail to increase in it to any marked extent on account of healthy blood having an unfavourable action on them. Consequently they disappear from it and become deposited in the internal organs, especially in the spleen, lungs, and liver, where they freely multiply. It is only after the anthrax vegetation has made a certain amount of progress in these internal organs that the blood loses its germicidal properties and becomes a good nutrient material for the bacilli, which then increase enormously in that fluid (Frank and Lubarsch). According to the experiments of Rodet, the bacilli are to be found in the blood before the first hour

after inoculation. The bacilli are most numerous in the capillary regions of the internal organs, as in the spleen, intestinal mucous membrane, mesentery, mediastinum, and lungs. For this reason, one often fails to find them in the blood of the peripheral and larger vessels. The enormous accumulation of bacilli in the capillaries frequently leads to laceration of these vessels and to extravasation of blood. Metastases of large accumulations of bacilli produce carbuncles and œdema in the internal organs and skin, accompanied by gelatinous extravasations and immigration of white blood corpuscles.

**The effect of the bacilli in the blood** is a subject about which little is known. Bollinger's mechanical theory of capillary engorgement and the theory of the deprivation of oxygen certainly explain the occurrence of hæmorrhages, but not the other features of the disease. The mechanical theory was directly opposed to the chemical theory, which asserted that the products of the metabolism of the bacilli, and not the bacilli themselves, were the cause of the symptoms of anthrax. Many objections have been raised against this chemical theory. Klebs, Pasteur, Nencki, and others, having separated the bacilli from their nutrient fluid by means of filtration through porcelain and with the aid of an air pump, have always found that the fluid itself was not virulent. In no case has anthrax been produced in animals by the inoculation of blood, or of liver or spleen tissue, or of material obtained from the foetus of a mother which was suffering from anthrax. In more recent times, however, Hoffa prepared nearly pure products of anthrax bacilli, and found these products to be extremely virulent, chemical, basic bodies which, as he states, produced in animals the symptoms of anthrax. He terms them anthrax-alkaloids (*Anthracin*) and considers that they are allied to the *neurin* of Brieger's ptomaines. Hoffa considers that the poisonous action of these anthrax-alkaloids and not the abstraction of oxygen from the blood by the bacilli, or the mechanical blocking up of the capillaries, is the true cause of death.

The effect of the virus of anthrax on the animal body seems to be favoured by certain conditions, especially hunger, weakness, fatigue, over-exertion and youth. For instance, Straus found by his inoculation experiments that puppies were much more susceptible to infection than old dogs, or full-grown guinea-pigs. Charrin and Roger showed that fatigue increased the susceptibility of white rats to the infection. Canalis and Morgurgo found even that originally immune animals (pigeons) became susceptible under the influence of hunger.

**General Remarks on the Occurrence of Anthrax.**—Anthrax attacks all animals; but most frequently cattle. The most susceptible animals are the herbivora (cattle, sheep, goats, horses, fallow deer, red deer, and camels), guinea-pigs, and mice. A single bacillus introduced into the subcutaneous connective tissue suffices to kill guinea-pigs and mice. Cats, tame rabbits, hares, and wild rabbits

come next in the order of susceptibility. Dogs, pigs, and foxes are very slightly susceptible. The supposed frequent cases of anthrax in pigs are mostly those of erysipelas which have been mistaken for anthrax. Rats, according to their breed, food, and individual idiosyncrasy, differ greatly among each other as regards their respective susceptibility to anthrax inoculation. Birds, with the exception of ducks, fowls, and pigeons, are immune. Fish and amphibia, especially frogs, very rarely suffer from anthrax. Foreign animals as a rule become easier infected than acclimatised animals, and well nourished ones than those in poor condition, especially at the commencement of an epizootic. One attack confers in some animals a certain amount of immunity.

Anthrax can occur sporadically, enzootically, and even epizootically. Most frequently it is a stationary disease restricted to certain districts. Its occurrence depends to a great extent on certain conditions of soil, vegetation, humidity, and temperature. Anthrax prefers black, loose, warm earth; mould, chalk, marl, and clay; soils which contain much organic detritus; and boggy, swampy, peaty, or brittle ground with an impermeable subsoil. The development of the bacilli is favoured by manuring with minerals, like lime and gypsum; and organic manures, such as stable-dung, compost, and mud from ponds (Nocard). There exists a well-marked connection between the disease and the amount of moisture in the ground. Anthrax likes a certain change in the moisture of the soil, and develops best on a moist, swampy, boggy soil which has partly dried up by long-continued summer heat. Wald, Buhl, Reinelt, Friedrich, Soyka, and other observers have noticed an increase in the number of cases of anthrax with a decrease of the moisture in the ground. The same rule holds good for dry mould, which becomes specially suitable for the development of the bacilli and spores, when, after temporary inundations, heavy showers, or thunderstorms, great heat with a rapid decrease of the ground-moisture sets in. This is the reason that anthrax appears most frequently in low lands and plains which are exposed to inundations. The development of the bacilli, being favoured by heat, takes place chiefly during the summer from June to September; but becomes arrested during winter, at which season cases not unfrequently occur by means of spore-containing dry fodder.

**Geographical Distribution.**—Anthrax is distributed

all over the world and in nearly every country. In Germany it is chiefly met with in districts where the soil is boggy, largely composed of vegetable mould, and having an impermeable subsoil. The principal anthrax districts in France are Beauce (Eure-et-Loir and Loiret), the department of Seine-et-Marne, Sologne, Burgundy, Poitou, Guyenne, Languedoc, Forez, Lyonnais, Auvergne, Dauphiné, and Provence. In Russia, anthrax is found most frequently in Siberia, Astrakhan, Orenburg, Perm, Novgorod, St. Petersburg, Archangel, Finland, and Lapland. It occurs in Switzerland, Austria (Galicia and Bohemia), Italy, England, Spain, Turkey, Africa, America, the East Indies,\* Persia, China, etc.

The money loss from anthrax in cattle is often very great, especially during the so-called anthrax years. From 1872 to 1875 it amounted in Upper Bavaria to £15,000 (over 900 head of cattle and 40 horses). During 1874, in the single district of Potsdam (Prussia), 2,000 red-deer and fallow-deer died from it. The total loss in Prussia from 1878 to 1883 came to more than £75,000 (over 6,000 head of cattle, 3,000 sheep, 300 horses officially registered, not counting numerous cases which had not been reported). In France the annual loss from anthrax amounts to many millions of francs. In former times it was even still greater. Delafond states that in the thirties the annual loss in Beauce alone was 10,000,000 francs (about 5,000 cattle and 300,000 sheep). The department Seine-et-Marne has had since 1859 a yearly loss of about half a million of francs ; and the department of Aisne one of 120,000 francs. In the government of Pskow, during 1884, 4,000 horses, nearly 2,000 head of cattle, and 1,000 smaller domestic animals died from it (W. Koch) ; and in the government of Novgorod, from 1867 to 1868, nearly 40,000 horses, over 8,000 cows, 6,000 sheep, and 500 human beings (Grimm).

**Statistics of Epidemics.**—In Germany from 1886 to 1894 about 30,000 animals were attacked with an average mortality of from 96 to 97 per cent. Cattle (23,000) suffered most, and after them sheep (5,000). Cases in horses (1,000) and pigs (350) were comparatively rare. There were only 50 cases in goats. The losses in the respective years were as follows :—

\* Anthrax is fairly well distributed all over India ; but is particularly prevalent in Eastern Bengal (Assam and Cachar) and Manipur. Loodianah disease and Manipuri horse disease are local Indian synonyms for anthrax.—Tr.

Year.	Cattle.	Horses.	Sheep.	Pigs.	Goats.	Total.
1886 . .	2,255	88	542	52	2	2,939
1887 . .	1,977	61	444	30	4	2,516
1888 . .	2,060	49	286	39	3	2,437
1889 . .	2,276	57	622	50	5	3,010
1890 . .	2,537	72	485	26	5	3,125
1891 . .	2,738	69	434	8	8	3,257
1892 . .	3,009	92	561	30	5	3,697
1893 . .	3,010	142	591	27	14	3,784
1894 . .	3,031	373	204	83	8	3,699

Among 10,000 healthy animals we find on an average 1.4 cattle, 0.2 horses, 0.25 sheep, 0.03 pigs, and 0.02 goats infected.

Anthrax statistics of foreign states are not so reliable as those of Germany. In Austria, anthrax was particularly rife in Galicia, Lower Austria and Bohemia. In Hungary, during 1889, 2,974 cattle, 8,284 sheep, and 387 horses were attacked; and in France, 1,306 cattle and 2,909 sheep. Only 2,900 cases of anthrax were reported in Great Britain\* from 1886 to 1890.

**The general anatomical changes in Anthrax** are chiefly as follows :—

1. Hæmorrhages by which all the organs are often permeated.
2. Serous, gelatinous and hæmorrhagic infiltrations of the sub-serous (especially of the mesentery and mediastinum), submucous (intestines) and subcutaneous connective tissue.
3. Swelling of the spleen and parenchymatous inflammation of the large glands of the body (liver and kidneys).
4. A tar-like condition of the blood with poikilocytosis and leukocytosis.
5. The presence of the bacillus of anthrax which is found in all the organs, especially in the capillary blood of the internal

\* The following are statistics of anthrax in Great Britain :—

Years.	Animals Attacked.				Total.
	Cattle.	Sheep.	Pigs.	Horses.	
1893 . .	833	108	313	46	1,300
1894 . .	625	125	188	62	1,000
1895 . .	604	158	140	32	934
1896 . .	632	34	200	38	904
1897 . .	520	39	284	38	881

organs (spleen, intestines, and liver) and in the serous gelatinous infiltrations of the mesentery.

The individual organs show the following changes :—

The vessels of the *skin*, on skinning the animal, appear to be densely filled with dark blood. On closer examination, we may observe under the epidermis and in the tissue of the cutis, small hæmorrhages which in some cases raise the epidermis into the form of vesicles. In anthrax due to inoculation we find in the skin, circumscribed, firm nodules of the size of a lentil up to that of a bean, with partial necrosis of the thickened tissue (the carbuncles of anthrax). Where there is considerable œdema of anthrax, the skin suffers from wide-spread necrosis.

The *subcutaneous connective tissue* is permeated with blood centres the size of a lentil up to that of a shilling, and is of a slightly blood-red tinge. In the œdema of anthrax we find circumscribed or diffuse gelatinous effusions of a rather firm consistence and frequently of a very considerable extent, which vary in colour from orange-gellow to yellowish-brown, or are permeated with spotted and flat extravasations of blood. Also, purely sanious infiltrations occur. The neighbouring lymph glands are considerably enlarged, permeated with hæmorrhages and œdematously infiltrated. The gelatinous infiltrations in the subcutis on the neck and along the trachea down to the chest are very large.

The *muscular apparatus* of the body varies in colour from a dark brownish red to violet, is infiltrated with hæmorrhagic spots, and is very soft. The muscular tissue of the heart shows similar changes (parenchymatous myocarditis).

In the *cavities of the body* (abdomen, thorax, and pericardium) a sanious fluid is found in moderate quantities. Hæmorrhagic extravasations of different sizes are seen under the serous membranes and epicardium, and particularly on the mesentery and mediastinum. The subserous connective tissue, especially on the mesentery, anterior cavity of the mediastinum, and in the neighbourhood of the kidneys, is gelatinously infiltrated, on which account the neighbouring lymph glands are considerably swollen, filled with serum and permeated with hæmorrhages (mesenteric glands and mediastinal glands). The internal organs (spleen, liver, kidneys and lungs) contain a large quantity of blood. All the larger veins and the heart are filled with blood, and the surrounding tissues show sanious imbibition.

The *spleen* is considerably enlarged (from 2 to 5 times its

normal size), either uniformly or by prominent tumours. The pulp of the spleen is soft, more or less fluid, and stained dark red. The capsule of the spleen is frequently extravasated with blood, and is always very tense. Sometimes small raised vesicles with sanious contents are to be seen on its surface.

The *liver*, like the *kidneys*, is highly congested and appears somewhat enlarged. The parenchyma is infiltrated with blood-centres and the cells manifest various kinds of degeneration (parenchymatous hepatitis and nephritis). The portal lymph glands often appear enlarged, and the retroperitoneal (perirenal) tissue is infiltrated with a serous, gelatinous fluid. Also, the sub-peritoneal tissue on the intestines and on the abdominal walls is frequently gelatinously or hæmorrhagically infiltrated; and the peritoneum inflamed.

The nature of the lesions of the *intestinal canal* varies according as the disease is intestinal anthrax, or anthrax caused by inoculation. In cases of inoculation-anthrax, the intestine is frequently normal. In other cases there may be subserous hæmorrhages or swelling of the mesenteric glands. The principal changes in intestinal anthrax are always found in the small intestine, chiefly in the duodenum, and more rarely in the colon. In the slighter cases of intestinal anthrax the mucous membrane is affected sometimes by a circumscribed, sometimes by a diffuse swelling. It has red spots, is infiltrated with hæmorrhages and covered with erosions, especially on Peyer's patches and the solitary follicles. The bacilli of anthrax are often found in extraordinarily large numbers on the surface of the mucous membrane. Necrosis and ulcers become developed in those parts where the bacilli most thickly congregate. In very severe cases we often find already on the abomasum or on the three first stomachs, gelatinous and sanious infiltrations of the mucous membrane. The mucous membrane of the abomasum, and especially of the duodenum, is, in consequence of excessive hyperæmia, dark red or almost black, and is covered with erosions and ulcers or necrosis, which may extend down to the sub-mucosa. The contents of the intestine are bloody, and the submucosa is infiltrated with a serous, gelatinous, or hæmorrhagic transudate, so that the mucous membrane often projects in the form of large tumours into the lumen of the intestine (œdema of anthrax). On the site of Peyer's patches and solitary follicles, we find flat or prominent nodules (carbuncles), the surface of which is covered with diphtheritic crusts and bacilli. The mesenteric glands are at the same time greatly enlarged



and hæmorrhagically and œdematously infiltrated. We seldom find the described lesion in the rectum except when its mucous membrane has become infected on account of its having been cleaned out with infected hands (carbuncle of the rectum). In this case the mucous membrane is swollen into thick prominences, and is partly necrotic and its surface is covered with blood.

Among the *organs of breathing* the lungs are greatly congested, œdematously infiltrated, and show ecchymoses in some places. The entire respiratory mucous membrane is considerably reddened and affected by ecchymoses. In particular, the mucous membrane of the pharynx and opening of the larynx is often so œdematously or gelatinously infiltrated (angina of anthrax), that excessive stenosis of the larynx takes place. The contents of the trachea and the bronchi consist mostly of bloody froth or mucus.

The *brain* is full of blood and studded with ecchymoses, and the surface of its membranes often exhibit hæmorrhages with an accumulation of sanious serum in the ventricles. Extravasations of blood sometimes occur in the anterior chamber of the eye and under the retina. All the other organs (the sexual organs, urinary organs, salivary glands, thyroid glands, bones, articulations, etc.) show hæmorrhages, and the urine frequently contains blood.

The *blood* is dark-red or almost black, has a tarry or varnish-like lustre, is of a watery consistence, and shows very little tendency to coagulate. It does not assume its normal red colour when exposed to the air. The red blood corpuscles appear in various stages of disintegration, and consequently their forms are very irregular (*poikilocytosis*). The number of the leucocytes is considerably increased (*leucocytosis*). Between the blood corpuscles we find, especially in the blood of the spleen, a considerable number of anthrax bacilli. The bodies of animals which have died from anthrax are often well nourished; they remain limp for a long time; grow cold very slowly (absence of rigor mortis); become quickly decomposed; and, as a rule, become greatly distended with gas. The visible mucous membranes are of a purple colour; blood flows from the natural openings of the body (mouth, nose and anus); and the rectum is sometimes prolapsed.

All the foregoing lesions may be absent in very acute cases (apoplectic form of anthrax). The characteristic bacilli of anthrax are, however, always present in the cadaver.

**General Symptoms of Anthrax.**—The symptoms of anthrax vary not only in the different species of animals, but also in individual cases, according as the intestinal canal, skin, lungs, etc., are respectively infected. It is even possible that the symptoms of intestinal anthrax may be quite different from the usual ones of this variety of the disease; or symptoms of metastatic affections of the intestine or serous membranes may become prominent in anthrax of the skin. The nature of the affection may vary essentially according to the amount of the virus of anthrax received by the animal body. Abortive cases and remittent forms of anthrax may also occur.

The characteristic features of all these forms of anthrax are the suddenness of the attack; severity of its course, which usually ends in death in from 1 to 3 days; grave general disturbance; high fever; tendency to hæmorrhage of the mucous membranes; and the presence of the bacilli in the blood. To these we must add the different local symptoms, such as carbuncles and oedema of the skin; intestinal trouble; brain complications; and difficulty in breathing. Anthrax has been classified according to its course (*anthrax acutissimus*, *acutus*, and *subacutus*), or according to the presence or absence of local manifestations. We consider the latter classification to be more appropriate than the former for clinical purposes.

### **Forms of Anthrax without Visible Localisation.**

—These forms, which can be divided into peracute, acute, and subacute, are generally due to ordinary infection by spores or to experimental feeding with spores.

1. The *peracute form* is known as *apoplectic anthrax*, and gives rise to symptoms of cerebral apoplexy. The animal becomes suddenly ill, staggers, falls down, often gives vent to a bloody discharge from mouth, nose, and anus, and dies in convulsions after a period of illness varying from a few minutes to 1 hour at the most. Frequently the patient is found dead in its stall in the morning, or may die suddenly while at work, pasture, or feeding. This form occurs most frequently in sheep and cattle, and particularly at the commencement of an epizootic.

2. The *acute form* runs a somewhat slower course and lasts from 2 to 12 hours, the maximum being 24 hours. The animal soon becomes feverish (40° to 42° C.) with an extremely rapid rise of temperature, and shows either signs of congestion of the brain or of congestion of the lungs. In the

former case, the chief symptoms are : restlessness, excitement, stamping, kicking, rearing, bellowing, attacks of fury, running to and fro, convulsions, spasms, grinding of the teeth, followed by stupor, insensibility, staggering, and finally death as if from apoplexy. In the latter case, the usual symptoms are greatly accelerated and difficult breathing, wheezing, panting, groaning, palpitations of the heart, excessively frequent and very small or imperceptible pulse, cyanosis of the mucous membranes of the head, bloody discharges from the natural openings of the body, hæmaturia, staggering, convulsions, and finally death from suffocation. Sometimes there is a partial remission of the symptoms with a relapse after a short time (*anthrax remittens*). In some cases we observe, before the commencement of the stormy course of the disease, premonitory symptoms of diminished vivacity, slight disturbances in digestion, constipation, tenesmus, etc.

3. The *subacute form* is termed *anthrax fever* or intermittent anthrax, and is the ordinary kind which attacks horses and cattle. The symptoms are generally the same as those of the acute form, except that they are more clearly defined and the course is longer ; 24 to 48 hours being the average duration, and 5 to 7 days the maximum. The phenomena of fever (rigors, unequally distributed temperature, and general disturbance of health) and those of congestion of the lungs and brain are more clearly marked, and are frequently complicated with severe intestinal troubles, especially colic. They may abate so frequently that the disease may be erroneously supposed to be of an intermittent nature.

**Forms of anthrax with visible localisation** appear to arise usually from infection by bacilli, as they can be experimentally produced in the form of carbuncles by inoculation.

The *carbuncles and œdema of the skin*, which are known as "*carbuncle disease*," chiefly occur in horses and cattle, and may also appear in dogs. The carbuncles are circumscribed cutaneous swellings, which are at first hard, hot, and painful ; and later on, cold, painless, and tending to become gangrenous. The œdema of anthrax consists of diffuse, flat, doughy, cold, painless, and frequently fluctuating swellings of the skin. The duration of the disease is from 3 to 7 days. Recoveries are more frequent than in cases of anthrax without external manifestations. Fever may appear before or after the formation of the swellings.

*Carbuncles and œdema of the mucous membranes* are mostly found on the mucous membrane of the mouth (anthrax of the tongue), in the pharynx and larynx (*anthrax angina*), and in the rectum. At the same time the patient suffers from fever, dyspnœa, laryngeal stenosis, difficulty in swallowing, general cyanosis, swellings in the intermaxillary space, and on the throat and the chest, painful straining to defecate, etc. Death takes place in from 12 to 24 hours. This form of anthrax is most frequent in pigs and dogs.

The usual forms in cattle are : " apoplectic anthrax " (p. 334), anthrax fever (p. 335), and " carbuncle disease " (p. 335) ; in horses, the same forms, and especially anthrax fever ; in sheep, apoplectic anthrax ; in dogs, carbuncles ; and in pigs, anthrax angina and anthrax of the tongue. We should be cautious in accepting supposed cases of anthrax in pigs.

**General Diagnosis.**—In the acute or subacute form, a correct diagnosis during life can be made only by a microscopical search for the bacilli, or by the knowledge of the epizootic character of the disease. A bacteriological examination of the blood very frequently gives a negative result, because the bacilli chiefly congregate in the internal organs. It has consequently been proposed to examine the blood of the liver after puncturing that organ with a trocar and cannula. The difficulty of the diagnosis *intra vitam* is further increased by the fact that the symptoms of the disease somewhat resemble those of simple congestion of the lungs or brain (although anthrax is to be distinguished from them by the presence of high fever), poisoning, rapidly appearing septicæmia, etc. Consequently an exact diagnosis can, as a rule, be arrived at only after death. The demonstration of the bacilli is generally necessary for the anatomical diagnosis ; for the microscopical result is rarely sufficient by itself. Johne recommends the following method for the detection of the bacilli : the cover glass preparations, which have been prepared in the usual way, and have been drawn three times through the flame, are stained for about a quarter of a minute (at the utmost for half a minute) by dropping a 2 per cent. watery solution of gentian violet on the cover glass, which must be cautiously warmed, along with the solution, washed for a moment in pure water, and afterwards for 8 or 10 seconds in a 2 per cent. watery solution of acetic acid, and finally very carefully washed again in pure water. A drop of water is then applied and the preparation examined with a magnifying power of at least 400 times. Klett advises

that the cover glass with the blood spread out on it should be allowed to dry in the air for some hours, drawn 3 times through the flame, stained for a few seconds with a watery solution of gentian and rinsed in water. After this, distilled water is dripped on the stained side of the cover glass, which is once more warmed by being passed from 6 to 12 times through the flame. It is rinsed again, placed on the object bearer, and examined when the upper side has become dry. By both methods the characteristic capsule of the bacillus of anthrax is made distinctly visible.

Inoculation is another aid to diagnosis; the most suitable animals being mice, rabbits and guinea-pigs. Sheep, if available, are very good subjects for inoculation. The diagnosis is confirmed by the occurrence of death after 2 or 3 days, and by the not very difficult demonstration of the bacilli in the cadaver. We may diagnose intestinal anthrax by inoculation with the fæces of the infected animals. An ordinary small wound in the skin will suffice for inoculation with the suspected material (blood, mucus, fæces, etc.). In some cases, inoculation with material from affected animals is unsuccessful, as for instance with blood from the surface of the body or larger blood-vessels which contain no bacilli; when the inoculation material is mixed with other bacteria; or when it is in a state of decomposition. All kinds of animals are not equally suitable for inoculation. Even in the same species of animals there may exist great differences of comparative immunity owing to breed or individual idiosyncrasy. Thus, very old animals are much less susceptible than very young ones. The technique of the inoculation, notwithstanding its great simplicity, may be defective, as when the introduced virus has been removed by the out-flowing blood, etc. Several young animals and, if possible, different species of animals should be inoculated at the same time.

The demonstration of anthrax is easier when a complete *post mortem* examination can be carried out than when only certain portions of the body are available.

1. If, after making a complete *post mortem* examination, we find the usual changes of anthrax in the large organs, and especially in the spleen, we shall be warranted in suspecting that anthrax was the cause of death. The correctness of this suspicion should be tested by an examination of samples of blood or of the spleen for bacilli, which we can do sufficiently well for the purpose by means of an ordinary microscope. We can easily make cover glass preparations.

2. If only single pieces of flesh are at our disposal, we can examine them for the presence of bacilli only by a very fine and most expensive

microscope, which few veterinary surgeons can afford to buy. We should remember that, besides the bacilli of anthrax, the affected flesh contains many other kinds of bacilli which cannot be distinguished from each other with an ordinary microscope. In order to avoid mistaking the bacilli of anthrax for harmless bacilli, we should make inoculations and subsequent bacteriological examinations, in rabbits and white mice for instance, or should prepare pure cultivations.

**Differential Diagnosis in General.**—As the various diseases which may be mistaken for anthrax will be discussed in detail when referring to the respective species of animals, we shall here content ourselves by giving the following brief description of those bacilli which possess some similarity to the bacilli of anthrax.

1. The *septic bacilli*, or the *bacilli of decomposition* (especially *bacterium termo*) are distinguished from the bacilli of anthrax, usually by their being motile, and by their rounded ends. Other bacilli which closely resemble the bacilli of anthrax, and can be distinguished from them only with difficulty, are sometimes met with. In such cases, the peculiar morphological relations, especially the presence of the capsule of the bacilli of anthrax, must be looked for, or test inoculations must be made.

2. The *bacillus of quarter-ill* is shorter and stouter than that of anthrax; the ends are rounded; and the bacillus is very motile, forms gas, and produces in cultivations variously folded dirty-white membranes. Being an anærobic fungus it cannot be cultivated on potatoes.

3. The *bacillus of malignant œdema* is thinner than that of anthrax; it is not articulated, and has rounded, not cut-off ends; it does not occur in the blood; it usually possesses slowly rocking movements; and grows out into very long and, frequently, circularly arranged threads, which are free from spores, and produces bubbles of gas in the cultures. A piece of tissue containing the bacilli of œdema placed in gelatine, forms around itself a white globe of fluid, the surface of which seems to be covered with fine needles. This bacillus can be cultivated only with great difficulty in laboratories (Kitt).

4. The *bacillus subtilis* (hay bacillus) is thick and short, shows oscillatory movements, and has a stout flagellum at both ends. This bacillus develops from a spore perpendicular to its longitudinal axis. Although it is ærobic, it is of much less importance in the differential diagnosis of anthrax than the other fungi we have mentioned (Kitt).

**Prognosis in General** is very unfavourable; the mortality being on an average from 70 to 90 per cent., and 100 per cent. in apoplectiform anthrax. After the epizootic has continued for some time, the disease sometimes assumes a milder type. Some animals acquire immunity for a short time from a previous attack. Recoveries without any treatment are not very rare.

**Therapeutics in General.**—Prophylaxis, which is much more important than treatment in cases of anthrax, consists first of all in the thorough destruction of the anthrax cadaver, and in the careful disinfection of the contaminated stalls. The destruction of the cadaver is best carried out by burning, when possible, or by burying it at least at a depth of 2 metres, and at some distance from roads, houses and pastures. It may also be rendered harmless by steaming under pressure sufficient to break up the soft parts. For disinfection we may use corrosive sublimate (1 to 1,000), creolin or lysol (3 per cent.), liquor chlori (2 per cent.), or chloride of lime (1 to 3).

An attempt to reduce the infectiousness of anthrax stricken districts should be made by draining swampy and damp ground, regulating the water supply, and keeping animals away from suspected pastures and suspected wells. A change of habitation can seldom be carried out as easily as one of food and water. The medical treatment, which is not always successful, consists in the internal application of hydrochloric acid, creolin, carbolic acid, or salicylic acid. Medicines are not of very much use; although almost every agent in the pharmacopœa has been tried. Meier reports instantaneous improvement and quick recovery in three cases of anthrax by the administration of 50 to 200 grammes of creolin. The following agents have also been recommended: corrosive sublimate (subcutaneous injection); carbolic acid and salicylic acid (10 to 15 grammes or more *per os* in cattle); iodine in the form of Lugol's solution, namely: iodine 30 grammes, iodide of potassium 60 grammes, distilled water 360 grammes, of which 2 tablespoonfuls in a quart of water is to be given every 2 hours; chlorine water; arsenic; phosphorus; hydrochloric acid; liquor ammoniæ; oil of turpentine; etc. It is customary in the subcutaneous form of the disease to administer neutral salts and other aperients, and small doses of calomel. In congestion of the lungs and brain, phlebotomy deserves a trial. The carbuncles of anthrax on the skin can be treated by long and deep incisions with

subsequent application of disinfecting remedies, or the actual cautery.

**Immunity against Anthrax, or Curative Inoculation.**—

According to the investigations of Ogata and Jasuhara, the protective and curative effect which has been obtained in certain infective diseases (tetanus, erysipelas, and human diphtheria) from blood serum taken from patients that had suffered respectively from these diseases, may also be obtained in anthrax. These observers state that even the blood of animals which are immune to anthrax (dogs, white rats, and frogs) is able to weaken the virulence of the bacilli of anthrax outside the animal body. Ogata extracted from the blood of dogs by means of alcohol and ether a ferment-like substance which had disinfecting properties, and which rendered animals immune to anthrax. As Enderlen, Kitt, and Grabitschewsky were not able to confirm these observations; we must wait for further results in serum-therapeutics. Emmerich, on the strength of his experiments with rabbits, recommends at an outbreak of anthrax, prophylactic inoculation of cattle, sheep, and other animals, with attenuated cultures of erysipelas. He states that the cocci of erysipelas increase the activity of the cells in an anthrax-infected organism to such a degree, that the tissues can successfully combat the bacilli of anthrax. Pawlowsky says that the coccus of pneumonia, the *staphylococcus aureus* and the *bacillus prodigiosus* have a similar effect.

**Protective Inoculation against Anthrax.**—There are three forms of immunity to anthrax, namely, acquired immunity from having successfully passed through a previous attack; racial immunity, as in the sheep of Algiers and Barbary; and individual immunity. On the other hand, it is impossible to render guinea-pigs, rabbits, rats or mice immune (Löffler); and horses can be made immune only with difficulty. Œmler produced anthrax in the same horse 7 times consecutively by inoculation. Man possesses no immunity. We can, by inoculation, produce in sheep and cattle an immunity which is, however, only partial and lasts for only a short time.

Toussaint was the first to make use of protective inoculations in anthrax. He heated anthrax blood which was free from fibrin, up to a temperature of 50° to 55° C. for 10 to 15 minutes, and used it directly afterwards as an inoculation material, although he had no true conception of the causes of its immunising action. Pasteur, however, was the first to prove that immunity was obtained by attenuation of the bacilli. The virulence of the bacilli of anthrax may be reduced by various means, such as heat (Pasteur, Toussaint, and Chauveau); compressed oxygen (Chauveau and Wosnessenski); antiseptics (Chamberland and Roux); and sunlight (Arloing). It can also be weakened by cultivating the bacilli in the blood of inoculated



sheep (Metschnikoff), or in the body of frogs (Lubarsch). Pasteur produced his inoculation material (vaccine) by the cultivation of bacilli at  $42^{\circ}$  to  $43^{\circ}$  C. in oxygen. He obtained his weak vaccine (*premier vaccin*) by a cultivation at the above-mentioned temperature continued for 24 days; and a second and stronger vaccine (*second vaccin*) by a cultivation continued for 12 days. The animals are at first inoculated with the *premier vaccin*, and after, from 10 to 14 days, with the *second vaccin*. The results of hundreds of thousands of inoculations *à la* Pasteur, which have been collected from almost every country in Europe (France, Russia, Hungary, Germany, Italy, Holland, and Belgium), vary to an extraordinary extent, and may be summed up as follows :—

1. Pasteur's protective inoculation is not to be recommended for *sheep*; because it produces in them either no immunity or an immunity which does not last more than a year. Consequently, to be efficacious, the inoculation has to be repeated annually. The mortality due to the inoculation is frequently very serious. In fact, it amounted in some cases, at the second inoculation, to from 10 to 15 per cent. Besides this, Pasteur's inoculation material varies greatly in strength, according as the temperature approaches  $42^{\circ}$  or  $43^{\circ}$  C. Koch states that, before use, Pasteur's *premier vaccin* should always be proved to be capable of killing mice, but not guinea-pigs; and the *second vaccin*, mice and guinea-pigs, but not rabbits. Sometimes the attenuated inoculation material, especially when it has been prepared too rapidly at a high temperature, shows a return to virulence; and on the other hand, may entirely lose its effect by being kept too long. A special kind of inoculation material is necessary for sheep, the different breeds of which are very unequally sensitive to the same kind of inoculation material. Lastly, we must consider the not insignificant cost of £7 10s. for inoculating 1,000 sheep with Pasteur's vaccine. The not very encouraging results obtained from the vaccination of sheep in France and Hungary show that, if protective inoculation be employed, it should be used only in districts where the disease is stationary, and where there are continued and heavy losses from it (over 2 per cent.), and when a good inoculation material is available.

2. Protective inoculations are of more practical value in *cattle* than in sheep, and can be recommended for use in anthrax districts. Individual animals vary greatly as regards immunity, which does not last longer in cattle than a year. The mortality is much less than in sheep; in fact, is almost *nil*. In them,

the inoculation as a rule causes only temporary fever and slight general disturbance. The vaccine of Chauveau and Perroncito is to be preferred for cattle to that of Pasteur, which is too weak. Although the former produces more severe general disturbance, it gives better results, and has the advantage that the act of inoculation can be performed in one operation. We may expect that with improved technique, the protective inoculation of cattle will become a very valuable supplement to the sanitary police regulations for suppressing anthrax.

**Technique of the Inoculation.**—The technique of Pasteur's method is as follows: The tubes coming from Pasteur's laboratory in Stuttgart or from Boutroux, 28 rue Vaucuelin, Paris, and costing £1 for fifty cattle or one hundred sheep, must be used at once and at one sitting, and must be opened only immediately before the inoculation, for which we use a Gramm's syringe. After the syringe has been well cleaned and disinfected before each application, the lymph tube which contains the *premier vaccin* is well shaken, the plug taken out, and the syringe filled directly from the tube. One eighth of the contents is subcutaneously injected on the inner side of the right thigh of the sheep, and the puncture is closed with the thumb. After 12 to 14 days the second inoculation is performed in the same way with the *second vaccin*. In cattle, a quarter of the contents of the syringe is injected behind the right shoulder with the *premier vaccin* and behind the left shoulder with the *second vaccin*. The hair should be removed from the site of the inoculation before the injection is made. Lambs, calves, and cows advanced in pregnancy should not be inoculated.

Chauveau has lately introduced a method for the rapid preparation of Pasteur's vaccine by heating the cultivations for 10 hours at 43° C.; for 3 hours at from 47° to 49° C., after renewal of the nutrient material; for 2 days at from 35° to 37° C. (for the formation of spores); and finally he raises the temperature to 80° C. He has also proposed attenuation by compressed air. Chamberland and Roux weaken the bacilli by chemical agents, as for instance, by the action of carbolic acid (1 to 600) for 24 days, or of bichromate of potash (1 to 2,000–5,000) for 10 days. Gibier makes his inoculation material by reducing the temperature of the bacilli to –45° C. Kitt attenuated the bacilli by passing them through the bodies of pigeons. Chauveau and Perroncito have made vaccines respectively for cattle and sheep by only one (instead of two as hitherto done) heating of the bacilli at from 37° to 38° C. for 5 days. According to Chauveau's latest method, the point of the glass tube containing the vaccine is broken off, and one drop for each animal is injected with a Pravaz syringe on the upper surface of the cartilage of the ear. According to Arloing, Cornevin and Thomas, the attenuated virus of anthrax may be restored to its former strength by the addition of lactic acid. Nocard and Roux consider that this increase of virulency is not due to any influence on the bacilli, but to a reduction of the power of resistance of the tissue cells by the action of the lactic acid.

**Statistics of Inoculation.**—The following statistics relate to the most

important protective inoculation experiments which have been carried out during the last few years :—

1. In *France*, which is the fatherland of anthrax inoculation, during the years 1882–1893 not less than 3,296,815 sheep and 438,824 cattle were inoculated. Out of that number, reports were received concerning the results of 1 $\frac{3}{4}$  million cases of sheep, and 200,000 cases of cattle. The mortality in the latter amounted to 0.34 per cent. ; and in the former to 0.94 per cent. Before the introduction of inoculation, 5 per cent. of all the cattle and 10 per cent. of all the sheep were said to have died from anthrax. The financial gain from inoculation is estimated in cattle at £80,000, and in sheep at £200,000.

2. In *Austria* 350,000 sheep and 130,000 cattle were inoculated *à la* Pasteur during the years 1882–1887, with a decrease in mortality, it is stated, of from 10 to 14 per cent. to from 1 to 2 per cent.

3. In *Hungary*, from 1889 to 1893, there were inoculated 87,430 cattle and 388,743 sheep, out of which 0.14 per cent. of the former, and 1.28 per cent. of the latter, died from anthrax.

4. In *Russia* 20,000 sheep were inoculated in the district of Cherson in 1888 (Skadowski), with a loss which was due to the inoculation, of 0.5 per cent. of the inoculated sheep, and 0.3 per cent. subsequently died from ordinary anthrax ; making a total loss of 0.8 per cent. It is stated that the loss from anthrax among non-inoculated sheep amounted to from 10 to 33 per cent. ; and that ewes inoculated after the third month of pregnancy produced immune lambs. The loss due to the inoculation in cattle (155 head) was 0.75 per cent. Skadowski recommends the adoption of protective inoculation in all places where the annual loss from natural anthrax amounts to 2 per cent. or more. In the Crimea, 4,564 sheep were inoculated in 1888 with vaccine obtained from Odessa (Bardach), the result being that 3,478 (80 per cent.) sheep died owing to a mistake having been made between the *premier* and *second vaccin*. Inoculations made with Pasteur's vaccine produced similar unfavourable results in the district of Odessa (Gamaleia). Wysokowicz states that in the district of Cherson the loss due to inoculation *à la* Pasteur amounted at first to nearly 2 per cent. in 1885 ; but from that year to 1888, out of 20,000 sheep which had been inoculated, only 170 (0.87 per cent.) died. According to Woronzow 1.25 per cent. of 7,000 inoculated sheep died in 1890 ; but none out of 200 head of cattle. It is stated that the previous annual loss was from 10 to 12 per cent. Podmolino reports that during 1892 and 1893, 67,000 sheep were inoculated in the district of Cherson with a loss of 0.43 per cent. ; 1,450 horses with a loss of 8 animals ; and 3,652 cattle, with a loss of 2. According to Ekkert, 38,936 sheep were inoculated in Southern Russia from 1883 to 1889 with a loss of 0.53 per cent. In the year 1890 the loss varied in inoculated adult sheep from 0.03 to 0.3 per cent. ; in lambs, from 0.1 to 3.6 per cent. ; and in particularly well-bred lambs the loss reached 13.3 per cent. ! The losses through natural anthrax fell in the inoculated flocks from 8–12 per cent. to about 0.1 per cent. Lange inoculated 928 animals without any dying ; and Gordsalkowski, 24,500 sheep with a loss of 0.3 per cent. Beresow states that in 1890–1891, 68,287 sheep were inoculated with a loss of from 0.3 to 0.8 per cent. ; 916 horses, with a loss of 0.3 per cent. ; and 1449 cattle without any casualty. Of the inoculated sheep, 2.2 per cent. died from natural anthrax after the inoculation.

5. During the years 1882-1888 in *Prussia*, Cœmler inoculated *à la* Pasteur 841 cattle and 3,459 sheep with a mortality in the former of 0.3 per cent.; and in the latter of 0.9 per cent. Subsequently 2.8 per cent. of the inoculated cattle and 1.9 per cent. of the inoculated sheep died of anthrax; consequently the total loss of the inoculated cattle was 3.1 per cent., and of the inoculated sheep, 2.8 per cent.

6. In *Italy*, that former zealous advocate of inoculation, Vicentini, now speaks unfavourably of it. In the province of Belluno, 18 out of 168 inoculated animals died. Other inoculations in 3,000 cattle caused decrease of milk, grave œdemata in a few cases, and some abortions and deaths.

7. In *Württemberg* 40 cattle were inoculated without any loss in 1891.

8. In *Holland* 255 cattle were inoculated during 1889-1890 with one death from anthrax.

9. The inoculation experiments which McFadyean made in *England* have convinced him that it would be inadvisable to introduce Pasteur's method into that country.

#### ANTHRAX IN CATTLE.

**Symptoms.**—Anthrax in cattle usually assumes the acute form without any outward manifestation. There is a sudden accession of high fever, with an internal temperature of 41° or 42° C.; the pulse is very frequent (80 to 100, or even more, in the minute), small, and scarcely perceptible; the visible mucous membranes of the head are greatly congested, and are frequently cyanotic; the conjunctiva is often infiltrated with hæmorrhages and considerably swollen; and the eyes are filled with tears. The external temperature of the body is unequally distributed; the hair stands on end; feeding and rumination are suspended; and there is great depression, weakness, stupor, and loss of sensation. The animal "gives way" in its hind-quarters; trembles over the whole body, especially about the flanks and pelvis; separates itself from the herd; assumes a staring, expressionless look; and manifests severe cerebral disturbance. Instead of stupor, attacks of madness and fury may come on; the animal may bellow, stamp its feet, rear up, and dash itself against any object it may encounter, etc. In other cases we may have symptoms of excessive dyspnoea without any apparent changes in the lungs. Gastric attacks may also supervene, as for instance, constipation, slight tympanitis, colic, and bloody diarrhoea, which are very common symptoms of anthrax in cattle, and which sometimes herald in the disease. The urine frequently contains blood (*hæmaturia*). Pregnant cows may abort or be attacked by strong labour-pains. A sanious discharge issues from the natural openings of the body (mouth,

nostrils, eyes, anus, and vagina), and death usually ensues in from 12 to 48 hours on account of general weakness and stupor, preceded by convulsions.

*Peracute Anthrax* (*anthrax acutissimus*) is observed, as a rule, sporadically in very strong individuals, and at the commencement of an outbreak. The patients die either quite suddenly, or in a few hours, with symptoms of cerebral apoplexy or poisoning. Previously healthy animals are sometimes found dead in their stalls in the morning.

*Subacute anthrax* is comparatively rare, and lasts from 3 to 7 days, or longer. It is distinguished by repeated remissions of fever and considerable emaciation.

The *carbuncles* of anthrax appear in cattle either primarily or during the course of the acute and subacute form, and affect different parts of the skin, such as that of the head, neck, chest, shoulders, abdomen, sheath, udder, flanks, or limbs. They are mostly isolated, circumscribed, or diffuse, and are little, if at all, painful, and then only at the beginning of the attack. They are of a blue-black or dirty dark-red colour, and when cut through are found to be gelatinous or lardaceous, but never suppurating. They become, however, often gangrenous, in which case there is deep gangrene of the skin. Carbuncles in the mucous membrane of the mouth (anthrax of the tongue, anthrax of the palate, and gloss anthrax) show themselves in the form of vesicles and nodes of varying size in the tongue and mucous membrane of the lips, cheeks, and palate with profuse salivation and difficulty of swallowing. Carbuncles on the mucous membrane of the rectum cause great straining during defecation, with swelling and prolapse of the mucous membrane, accompanied by a bloody discharge. In most cases the cause of this implication of the rectum is injury inflicted during back-raking.

**Differential Diagnosis of Bovine Anthrax.**—The diseases for which anthrax is most frequently mistaken are : poisoning, inflammation of the brain, cerebral apoplexy, pulmonary apoplexy, heat apoplexy, death by lightning, rabies, gastro-intestinal inflammation, leucæmia, foot-and-mouth disease, and quarter-ill. As the course of the disease is very rapid, it can often be recognised only, *post mortem*, by finding the bacilli and by employing test inoculations. With reference to the differentiation of quarter-ill see p. 121.

## ANTHRAX IN HORSES.

**Pathogenesis.**—Many of the reports on anthrax in horses are unreliable; because in them petechial fever has been frequently mistaken for anthrax. The disease generally occurs in anthrax districts, where nearly all the domestic animals may become infected. The absorption of anthrax spores or bacilli by means of the fodder is consequently regarded as the usual cause. Anthrax is very common among the horses of Asiatic and European Russia, where it occurs chiefly in swampy districts which are exposed to inundations, such as the low-lying countries of the Volga, where it is known as “Siberian plague” (Haupt and Blumberg). In German South-West African protected territories it goes under the name of “*Bloed-ziekte*,” *i.e.*, blood disease (Sander). The infection may be communicated along with the stings of flies. The disease may be transferred indirectly from cattle or sheep to horses in other ways, as for instance, by putting on harness of untanned infected leather (Bobertag).

The contagium may be introduced into perfectly healthy districts and stables by imported oats or hay, which theory is the only explanation that can account for the rare sporadic cases of anthrax in the army. In the whole Prussian army only 15 horses were affected with, and died from, anthrax in the years 1889 to 1894.

**Symptoms.**—Anthrax occurs among horses usually in the acute or subacute form. The first symptom is high fever, ranging from  $39.5^{\circ}$  to  $41.5^{\circ}$  C., or more, with a very small and finally imperceptible pulse of from 80 to 100 per minute. The rise in temperature is often manifested by rigors and muscular spasms. The external temperature of the body is very unequally distributed. The mucous membranes of the head, especially the conjunctiva, are cyanotic, and sometimes slightly yellow. Lacrymation of both eyes is often present, and the mental faculties are exceedingly torpid. The animal has a dull, stupid look, is apathetic, appears stunned, and staggers when walking; or there are symptoms of inflammation of the brain (excitement, restlessness, and convulsions). Colic, which frequently appears at the beginning of the attack, is a regular and very characteristic symptom of anthrax in horses. It is seldom very severe, and is followed by watery dysenteric diarrhoea. As in cattle, the respiration is exceedingly accelerated and difficult. In cases of

carbuncular swellings of the mucous membrane of the pharynx and the larynx, we find symptoms of angina such as salivation, difficulty in swallowing, large swellings in the region of the larynx and the pharynx, dyspnoea, and even suffocation. Death takes place with increasing weakness, profuse perspiration, etc., in from 6 to 30 hours, on an average. In a few cases the disease may run a fatal course in from a quarter to half-an-hour. Recoveries are very rare.

*Carbuncles on the skin* occur chiefly on the hypogastrium, lower part of the breast, inner surface of the fore-quarters and hind-quarters, scrotum, vulva, etc. The swellings of the hind-quarters cause lameness. This form of anthrax runs a course of 2 or 3 days. Gloss anthrax, namely, carbuncles in the mucous membrane of the tongue, have been found on rare occasions in horses (Gresswell).

[For convenience sake, we may divide equine anthrax into anthrax accompanied by swelling of the throat, and anthrax without this symptom. In the former, the disease seems to be located chiefly in the organs of breathing; in the latter, in those of the abdomen. The one might be termed the thoracic or respiratory form; the other, the abdominal. Fred. Smith (*Veterinarian*, February and March, 1897) states that the latter is about 4 times as common as the former in horses. In the cases I have seen, principally in Assam and Cachar (India), the respiratory variety was the more frequent. As addenda to the symptoms described in the text, I may state that the animal suffers from depression of spirits and stupor. The breathing (in the thoracic form), which is almost entirely abdominal, as may be seen by the heaving of the flanks, becomes more and more hurried, until at last the animal falls from exhaustion, becomes convulsed, and dies, apparently from suffocation. There is sometimes a flow of a rusty-coloured fluid from the nostrils, and often, towards the end, and after death, a discharge from them of foam more or less tinged with blood. I have never seen the disease kill in less than 3 or 4 hours from the first appearance of the symptoms. The course of the malady may run on for 6 or 7 days. The mortality from anthrax in India is probably about 80 per cent. When there is swelling of the neck, the nose is poked out, and the neck (especially about the region of the throat) and intermaxillary space become swollen, often to an enormous extent, so that the head and neck become hard and immovable, as if they were cut out of a single piece of wood. The amount of the swelling, however, greatly varies.

In the abdominal form of anthrax there is sometimes prolapse of the rectum; and, in a few cases, serous tumours form about the flanks, underpart of the belly, and scrotum.

To state the matter generally, the symptoms of anthrax are those of impeded respiration and of infiltration into various tissues. Hence, the painful breathing, the interference with the functions of the brain, the semi-paralysed condition of the patient, and the local swellings.—  
Tr.]

**Differential Diagnosis in Horses.**—Anthrax is distinguished chiefly by its stormy and feverish course, and by the variety and rapid changes of the different symptoms, which never appear in regular succession. It is confused most frequently with petechial fever, and next to that with colic, cerebral apoplexy, œdema of the lungs and septicæmia. As we have already mentioned when discussing petechial fever, this disease was frequently, even in recent times, regarded as a form of anthrax, which view is altogether wrong; for the bacilli of anthrax have not been found in the blood of the animals affected with petechial fever; and all attempts to transmit petechial fever, either by inoculation or by infection, have failed. A correct diagnosis of anthrax can be obtained only by the demonstration of the bacilli, and by transmitting the disease to a rabbit or a sheep by inoculation.

[On account of the hurried breathing and injected condition of the mucous membranes of the nostrils and eyes, anthrax is sometimes mistaken for congestion of the lungs, which can be distinguished by the fact that the patient fights for breath, and does not exhibit the depression and semi-unconsciousness of a horse suffering from anthrax. Besides, the characteristic swelling of the neck is absent.

The disease which, above all others, clinically resembles equine anthrax is South African Horse Sickness (p. 467), especially when the attack of anthrax takes the respiratory form. The clinical resemblance between the two is greatly heightened by the fact that the symptom of swelling of the head, intermaxillary space and neck is common to both. Sander (p. 421) is by no means alone in the mistake he made; for many English veterinary surgeons who have sojourned in South Africa have fallen into the same error. In fact the question was not finally settled until Edington had published the results of his bacteriological researches on the South African malady. Here, the most salient points in *differential diagnosis* are: Influence of time of the year on Horse Sickness. (2) Influence of dew on the development of the South African micro-organism. (3) Protection against Horse Sickness by restriction in diet to dry fodder. (4) Difference in the character of the nasal discharge; that of Horse Sickness being generally white and very frothy, with an extremely copious flow of liquor sanguinis. (5) Charged condition, in Horse Sickness, of the bronchial tubes with fluid. (6) Difference in the *post mortem* appearance of the blood. (7) Unchanged condition of the spleen in Horse Sickness. (8) The fact that Horse Sickness is peculiar to the equidæ. (9) Absence of the bacillus anthracis in Horse Sickness.—Tr.]

#### ANTHRAX IN SHEEP AND GOATS.

**Pathogenesis.**—Gerlach was the first to recognise as anthrax the disease of sheep which was formerly known as "apoplexy." It appears to be almost always due to fodder.



Stings of flies, injuries of the skin during shearing (Nocard), etc., may in some cases be the direct or indirect cause of the disease.

**Symptoms.**—Anthrax attacks *sheep* generally in the apoplectic form (*anthrax acutissimus*). The animal appears as if suddenly stricken with apoplexy, staggers, falls down, is seized with spasms and convulsions, and dies in a few minutes with a discharge of black blood from the natural openings of the body, and is often found dead in the stall in the morning. The course of acute anthrax lasts somewhat longer, although generally only from 30 minutes to 2 hours. We sometimes find symptoms of congestion of the brain, such as excitement, restlessness, running to and fro, staggering, etc., and at other times, symptoms of congestion of the lungs, namely, very accelerated breathing, frequent pulse, palpitation of the heart, cyanosis of the mucous membranes, bloody discharge from the natural openings of the body, etc.

Subacute anthrax is very rare in sheep. It is sometimes ushered in by premonitory symptoms, especially by digestive and intestinal troubles of an inflammatory nature. Frequent and strenuous attempts to dung, with constant whisking of the tail, is a characteristic symptom. Carbuncles are seen very rarely on the head, throat, or udder.

The course of anthrax in *goats* is similar to that in sheep; but is not so severe as a rule.

**Differential Diagnosis.**—Anthrax in sheep has been confused most frequently with malignant œdema. The affections which Haubner and others described as anthrax, and which were characterised by crackling swellings of the hind limbs, were certainly malignant œdema.\* In cases of doubt, Cadiot and Reis recommend that pressure should be made on the nose of the patient with the fingers for a few seconds, to excite urination. It is stated that hæmaturia is always present in anthrax. [Anthrax in sheep is often confused with braxy and loup-ill.—Tr.]

#### ANTHRAX IN PIGS.

**Pathogenesis.**—Swine erysipelas (p. 70), which was formerly almost universally regarded as a variety of anthrax, has

\* With respect to this statement, *The Journal of Comp. Path. and Therap.*, Sept., 1898, remarks that "it is far more probable that the cases referred to were quarter-evil."—Tr.

nothing to do with it. Consequently, the literary reports of former times on anthrax in pigs are of little value. Pigs are more or less immune to anthrax, which can be transmitted to them only with great difficulty. In fact, the inoculation experiments of Brauell, Renault, Toussaint, Arloing, Cornevin, Thomas, Schindelka, von Ratz, and others, have been attended with negative results. True cases of anthrax seem to occur among pigs only during widely-spread anthrax epizootics and in anthrax districts, or after eating the flesh of anthrax-infected animals,\* in which case local affections in the pharynx and mouth usually appear. These remarks are confirmed by the recent experiments of Crookshank, who has proved that anthrax can be produced in both old and young pigs by feeding with portions of the dead bodies of anthrax-infected animals. Trombitas saw 14 pigs die of anthrax within 6 days, after having been fed on the flesh of an anthrax-infected cow. Von Ratz observed in pigs of a Polish and Chinese cross that the anthrax infection took place at the beginning of the digestive canal, most probably from the follicles of the tonsils. According to his experience, the power of resistance to anthrax varies in different breeds of pigs. Crookshank and Perroncito succeeded in experimentally producing the disease by the injection of virulent blood and of bacilli cultures.

**Symptoms.**—Anthrax angina is characterised by carbuncles on the mucous membrane of the pharynx and larynx. The animals are very feverish, and the intermaxillary space is greatly swollen. This swelling may spread along the course of the trachea to the lower part of the chest and the inner surface of the fore-quarters, and thus gives rise to symptoms of angina of the pharynx and larynx, such as salivation, retching, stiff attitude of the head and neck, hoarseness, difficulty in swallowing, regurgitation, vomiting, cyanosis of the mucous membrane of the mouth, dyspnoea, panting, harsh breathing, etc. The skin is stained with blood, and the animal shows symptoms of paralysis. Death takes place usually from suffocation. In anthrax of the tongue and palate, clear vesicles, which subse-

\* McFadyean reports an outbreak in which about 14 pigs were attacked with anthrax, probably from eating the flesh of an anthrax-stricken heifer. In all of these pigs, the most prominent symptom was swelling in the region of the throat. McFadyean states that "swelling of the throat in the course of an attack of acute illness is in the pig almost pathognomonic of anthrax."—TR.

quently become purple and even black, are seen on the mucous membrane of the tongue, palate, lips, etc. Zschokke has described in pigs the occurrence of carbuncles on the skin of the back.

The anatomical changes in anthrax of pigs consist in a gelatinous, hæmorrhagic infiltration of the parts surrounding the pharynx; hæmorrhagic swelling of the lymph glands, spleen and liver (according to Crookshank, the spleen may be normal); and hyperæmia of the other organs. In some cases, enteritis with submucous and subserous hæmorrhages is present. Although the spleen and blood contain only a few anthrax bacilli, they are rich in putrefactive bacteria, the presence of which greatly increases the difficulty of the microscopical demonstration of the anthrax bacilli, and transmission by inoculation (Crookshank)

#### ANTHRAX IN DOGS AND CATS.

Our knowledge of anthrax in dogs is very defective. According to Straus, age confers on them comparative immunity. This observer states that newly-born dogs are extremely susceptible to anthrax when inoculated with moderately virulent material. Bardach inoculated anthrax intravenously in 25 dogs from which the spleen had been removed, and in 25 normal dogs. Of the former 19 died of anthrax; of the latter only 5. The eating of meat infected by anthrax seems to be the most frequent cause of the disease in these animals. On this account, anthrax occurs in dogs usually in the intestinal form and in that of local anthrax of the mouth and pharynx. Cornevin saw 5 dogs out of 7, which had been eating parts of the dead body of an anthrax-infected cow, die on the evening of the same day. Peuch saw a case of anthrax from feeding on infected flesh in a dog which had a wound on the tip of its tongue. Some supposed instances of anthrax in dogs are probably nothing else than cases of ptomaine poisoning, which is a disease that was formerly frequently confused with anthrax in man.

Cats, lions, and other carnivora may become infected with anthrax in a similar way to dogs.

#### ANTHRAX IN BIRDS.

**Pathogenesis.**—Pasteur states that birds are immune to anthrax on account of the high temperature of their blood; but that they may be made susceptible by reducing their bodily

heat. Pasteur's statements have lately been confirmed by Wagner, who found that this immunity is lost by the introduction of certain chemical agents (chloral hydrate and antipyrin) which have a weakening effect on the birds. Czaplewski also found in his experiments that only very young pigeons of certain breeds are susceptible to anthrax. Koch, Gaffky, Löffler, Perroncito, Hess, Kitt, and others, obtained negative results with their inoculation experiments in birds. On the other hand, the inoculation experiments of Feser and Emler have been successful in several cases. Emler observed in his experiments that small birds (red-breasts, finches, goldfinches, canaries, sparrows, yellow-hammers, etc.), and especially young birds, can be very easily infected; that the larger birds are less susceptible; and that only birds of prey are quite immune. Usually birds do not become infected, except during a general anthrax epizootic, and after feeding on flesh and blood of anthrax cadavers. Farmyard poultry may also become infected.

**Symptoms.**—The course of anthrax in birds is exceedingly rapid and severe, and may terminate suddenly or after a few hours' suffering. Towards the end, the bird staggers and trembles, or becomes violently convulsed, and dies with a bloody discharge from the mouth, nostrils, and anus. In other cases the disease lasts for about a day. The bird is depressed and weak, its feathers are ruffled, it droops its wings, and suffers from dyspnœa and bloody diarrhœa, and the mucous membranes and corpora cavernosa (comb and wattles) are livid. Carbuncles are comparatively frequent, and appear chiefly on the comb, wattles, conjunctiva, tongue, palate, extremities, webbed portion of the feet, etc. Mistakes are often made between anthrax and chicken cholera, cerebral apoplexy, and malignant œdema.

**Anthrax in Man** is chiefly found in knackers, butchers, tanners, herdsmen and veterinary surgeons, in consequence of an infection acquired whilst cutting up the cadavers, and it generally assumes the form of carbuncles (malignant pustule) on the arms, face, neck, etc. The appearance of the pustule is followed by fever, and usually by death. Intestinal anthrax mostly occurs from eating infected flesh. The treatment of malignant pustule consists in its destruction by the hot iron, excision or by the use of strong disinfectants, such as corrosive sublimate, creolin and carbolic acid.

RABIES (*Hydrophobia*).

Rabies in general—Canine rabies—Symptoms of rabies in man—Bovine rabies—Equine rabies—Rabies in cats—Rabies in pigs—Rabies of sheep and goats—Rabies of birds.

**History.**—Rabies is one of the oldest known diseases of animals. In the fourth century, B.C., it was described by Aristotle, who writes: "Dogs suffer from madness which puts them in a state of fury, and all the animals that they bite, when in this condition, become also attacked by rabies." We find allusions to it in the works of Virgil, Horace, Ovid, and Plutarch. Cornelius Celsus, who lived in the first century of the Christian era, was the first to allude to human rabies, and to employ the word "hydrophobia." Dioscorides recommends excision of the wound as a protective remedy; and Galen in the second century gives special remedies for rabies. Among the older authors we may cite Plinius the younger, Columella, and C. Aurelianus. No allusion to rabies is to be found in the literature of the Middle Ages. Bauhin, in 1591, mentions in his "*Memorabilis historia luporum aliquot rabidorum*" (the transmission of rabies by wolves to man). In 1604 an epizootic of rabies broke out in Paris (Andry); towards the end of the seventeenth century, in Italy (Baglio, Rammazzini); in 1708, through Swabia (Camerarius and Scharff); from 1719 to 1723, in France and Germany; from 1754 to 1760, in England; and from 1779 to 1807, in America, especially through the West Indies and Peru. Towards the end of the eighteenth, and at the beginning of the nineteenth century, rabies spread over the whole of Europe, and many rewards were offered during that time for the discovery of a remedy for it. The names of Chabert and Hunter should be kept in remembrance for the good work they did in the investigation of this disease. An epizootic of rabies raged amongst foxes from 1803 to 1830 in Southern Germany and Switzerland (Köchlin and Franque). During 1814 and 1815, Viborg in Copenhagen and Waldinger in Vienna improved the methods of the experimental investigation of rabies. In 1817 and 1818, Delabère Blaine and Greve in England greatly enriched the clinical knowledge of this malady. Rabies was prevalent during 1822 in Holland; during 1823 and 1824 in Berlin; and during 1824 in Sweden and Russia. In 1828, during the epizootic which raged from 1823 to 1830, Hertwig published his "Contributions to the

better understanding of Rabies," which is a report of a great number of carefully-executed experiments on the transmission of rabies, and which greatly advanced the knowledge of this disease. In 1832 Youatt in England and Prinz published their works on rabies. In the thirties the disease was very common in Prussia; and from 1838 to 1843 in Austria and Württemberg. During 1852 and 1853 Prussia was again visited by this scourge. In 1853, 150 dogs suffering from rabies were taken to the canine hospital of the veterinary college in Berlin, and 267 cases occurred in Hamburg. During 1861 rabies broke out as an epizootic in the Rhenish countries and in France; and from 1863 to 1871 in Württemberg, where 597 animals became infected, and 449 people were bitten, of whom 23 died. From 1862 to 1867, and again from 1873 to 1876, it raged in Vienna; from 1865 to 1866 in Saxony; and from 1871 to 1876 in Saxony, Bavaria, and Prussia. Since the introduction of the Imperial laws respecting epizootics, the number of cases of rabies has very considerably decreased in Germany.

Erroneous opinions about the nature of rabies have been held for a long time. In spite of the proved infectious character of the disease, it was formerly believed by the majority of people that rabies could originate from the respective influences of great heat, unsatisfied sexual desire, thirst, nervous excitement, anger, jealousy, too high feeding, etc. Virchow and others called attention as early as 1854 to the error of this supposition. It is only during the last few decades that the exclusively infective nature of the disease has been generally accepted. In the year 1881 Pasteur greatly increased our knowledge of the nature of this disease by his work in the field of protective inoculation, and gave a permanent impulse to further experimental researches.

**Etiology.**—The infective matter of rabies has not yet been produced in a pure condition in spite of the labours of numerous investigators, such as Raynaud, Lannelongue, Gibier, Fol, Babes, Pasteur, Koch, and others. Pasteur, however, has shown that the virus is purest in the central nervous system (brain and spinal cord) of infected animals, and less so in the peripheral nerves, salivary glands, lachrymal glands, aqueous humour of the eye, pancreas, mammæ, testicles, kidneys, and in their secretions. In the urine the infective matter is mixed up with numerous other micro-organisms. The nervous system appears to offer the most favourable condition for the develop-

ment of the virus, which does not seem to be contained in the blood. The contagium is at all events fixed, not volatile; and is purely endogenous, not octogenous; that is to say, the animal body is absolutely necessary for its development. Paul Bert proved that it is a solid body, by filtering the saliva of rabid dogs through plaster of Paris plates and by then showing that the filtrate was innocuous. Pasteur found microscopically in the brains of rabid animals fine granules in the form of extremely minute points which could be stained with aniline solutions, and which he regarded as very minute micro-organisms. He did not succeed in cultivating these bodies, which he considered to be neither micrococci nor bacilli.

The recent investigations of Babes show that the existence of a characteristic micro-organism of rabies cannot be proved even by our most modern bacteriological methods. These investigations, however, point to the probability of our being able to cultivate through several generations, from the central nervous system of rabid animals, an unknown substance which possesses the capability of producing rabies, and behaves to external agents, as a rule, like a bacterium; although it is much more resistant to carbolic acid than any known bacterium. Babes cultivated this substance and produced rabies in dogs, rabbits, guinea-pigs, cats, rats, and mice by inoculating it into the vitreous humour of the eye, or between the cerebral membranes by trephining. Anrep obtained from the brain of rabid rabbits an exceedingly virulent material which appeared to be of the nature of an alkaloid.

The vitality of the contagium of rabies seems to be greater than was formerly supposed. The inoculation experiments carried out by Hertwig by means of the saliva and blood of rabid dogs gave negative results 24 hours after their death. Gibier, on the contrary, found that the saliva remained virulent for 24 hours, and Mergel observed that the brain continued virulent for 15 days. Similar results were obtained in Dorpat. Pasteur preserved the brain of rabid dogs for 3 weeks under a very low temperature ( $-12^{\circ}$  C.) without the infective matter losing its virulence. This organ, if kept in moist carbolic gauze, retains the contagium in an active condition for months. The infective matter remained virulent for 3 or 4 weeks during summer, when kept in tubes which had their ends closed by fusion. Pasteur proved that the virus can retain its power of infection for 4 or 5 days when exposed to far advanced putrefaction. It was

formerly supposed that it lost its infective power directly after putrefaction set in, and consequently soon after the dead body had cooled down, that is to say, within 24 hours after death. Galtier found that, in spite of putrefaction, buried cadavers remained virulent for 15 to 44 days. According to Viala, the contagium of rabies continues active for over 5 months when kept cool and in a vacuum. Bruschettini states that it becomes inert after having been kept in carbonic acid for 13 days.

In all cases, the transmission of the infective matter to other animals appears to be directly effected by the bite of a rabid animal without any intermediate bearer. Rabies is therefore to be looked upon as an inoculation disease, the saliva serving as the vehicle for the contagium. Roux and Nocard have pointed out the very important fact that 2 or even 3 days before the appearance of rabies, the saliva contains the contagium, and is consequently virulent towards the end of the period of incubation. It is very doubtful if the virus can be absorbed in the digestive canal by the consumption of the flesh, milk, or saliva of rabid animals, as Galtier states was the case with rabid saliva in his experiments with rabbits. We may be quite certain that the ingestion of the flesh or milk of rabid animals has never produced any injurious consequences. As a proof of this fact we may mention that Nocard fed a young fox for 2 months on the brain and spinal cord of 12 rabid dogs, without any ill result to the fox. Wirchikowski states that gastric juice destroys the contagium of rabies. On the other hand, we have to point out that the intra-cranial inoculation of the milk of rabid animals has produced rabies (Nocard, Roux, and Bardach). Perroncito and Carita produced rabies in guinea-pigs by inoculation with the medulla of a young rabbit, the mother of which had become rabid by inoculation. Callignac and Gibier state that they have observed hereditary transmission of rabies. The experiments of Renault, Roux, Galtier, Zagari, Bombicci, and others on this point have, on the contrary, given negative results. In any case, the hereditary transmission of rabies is certainly very rare.

#### **The Behaviour of the Virus of Rabies with Various Agents.**

—According to the investigations of De Blasi and Russo-Travoli, the rabic virus is destroyed by a 1 per cent. solution of creolin and by lemon-juice in 3 minutes; by Ceylon cinnamon, a 2 per cent. solution of *liquor ferri*, a 5 per cent. solution of hydrochloric acid and salicylic acid, and a 50 per cent. solution of nitrate of silver, in 5 minutes; by



sulphuric acid and liquor ammoniæ in 10 minutes; by a 4 per cent. solution of boric acid in 15 minutes; by a 1 per cent. solution of permanganate of potash in 20 minutes; by a 5 per cent. solution of carbolic acid in 50 minutes; by a 3 per cent. solution of carbolic acid in 60 minutes; and by a 2 per cent. solution of carbolic acid in 120 minutes. According to this, creolin is the strongest and carbolic acid the weakest disinfectant for the virus of rabies. We learn from the experiments of Celli that the virus of rabies is rendered inert by hot steam in half-an-hour; by a temperature of from 50° to 60° C. in 1 hour; by one of 45° C. in 24 hours; by corrosive sublimate (1 to 100,000), permanganate of potash (from 2 to 5 per cent.) and alcohol (50 to 90 per cent.) in 24 hours; by a 25 per cent. solution of alcohol in 5 days; and by a 15 per cent. solution of alcohol in 7 days. The emulsion of the brain substance instantly loses its virulence when it is slightly acidulated with one or two drops of acetic acid, or when it is made slightly alkaline with soda.

**Pathogenesis.**—If the virus contained in the saliva of a rabid animal penetrates the skin, it may remain for a long time at the site of the bite, or it may enter sooner or later into the body by means of the blood or along the nerve-tracts. According to the most recent investigations, it appears that the virus of rabies moves from the bitten part chiefly within the nerve-tracts in a centripetal direction to the central nervous system, possibly enclosed in migratory cells (Duboué, Di Vestea, and Zagari). It is evident that the virus becomes best developed in the brain and spinal cord; the former being the principal seat for the development of furious madness; the latter for that of dumb madness. Inoculation experiments have shown that the disease breaks out most rapidly from direct inoculation into the brain, under the dura mater, and that the period of incubation is proportionate to the distance of the site of the inoculation from the brain.

The *period of incubation* is longer than in other infective diseases, and in dogs amounts on an average from 3 to 6 weeks, with a maximum of several months, and a minimum of only a few days. [Hunting puts the usual period at about 25 days.—Tr.] The virus may remain for a long time (as we have already said) at the site of the bite and may become absorbed only in small quantities at a time; or it may rapidly and in large quantities penetrate into the body. According to Protopopoff, the younger the animal, the shorter is the period of incubation.

**Occurrence.**—Although rabies chiefly attacks dogs, it may affect any of the higher animals, even birds. Out of all the

supposed predisposing causes—such as breed, age, sex, climate, country, etc.—the season of the year is the only one which has any influence. As in other infective diseases the number of cases of rabies are greater in summer than at other times. Some animals appear to possess individual immunity to it. It goes without saying that the disease is most frequent in places where there is a large number of dogs, and where there is much traffic, as in large towns, especially when the protective police measures are defective. On account of the disease being transmitted from dogs to wolves, and *vice versa*, it is frequently enzootic in the neighbourhood of mountains and plains in which wolves are found, as in the Vosges, Carpathian Mountains, Russia, etc.

**Statistics.**—In the German empire nearly 5,000 animals (dogs, cats, horses, cattle, sheep, pigs and goats) became rabid from 1886 to 1894. In Germany, on an average, about 400 dogs and 100 other animals annually become rabid. The figures for the individual years are as follows :—

Year.	Total number.	Dogs.	Cattle.	Horses.	Sheep.	Pigs.	Cats.	Goats.
1886 . . .	578	438	92	5	32	7	3	1
1887 . . .	556	423	99	6	6	17	4	1
1888 . . .	547	397	101	7	17	18	5	2
1889 . . .	493	410	65	5	3	6	4	—
1890 . . .	714	590	98	4	2	9	11	—
1891 . . .	542	445	70	11	8	4	3	1
1892 . . .	500	387	69	8	7	27	2	—
1893 . . .	466	410	39	3	7	4	3	—
1894 . . .	557	471	73	4	—	6	3	—
Total . . .	4,953	3,971	706	53	82	98	38	5

Every year about 1,000 animals become rabid in Austria; 2,500 dogs in France; 1,000 in Belgium; and about 900 in Great Britain [see note, p. 361]. It is stated that rabies does not occur in Australia, because a six-months' quarantine has been imposed on imported dogs. [South Africa is practically free from rabies. The only case reported for many years was one of a dog which was found to be rabid in Port Elizabeth, by Britton, in 1894].—TR.

**Anatomical Changes in Rabies.**—In contrast to other infective diseases, the changes which are found *post mortem* in rabid animals are neither constant nor specific; in

fact they are characterised more or less by negative appearances. The general changes, which are in no way typical, are as follows : The very emaciated cadavers become rapidly putrid, and, in the case of the larger domestic animals, greatly distended by gas in the hind parts of the body. The blood is thick and of a dark-red colour ; the muscles appear granular and affected with fatty degeneration, and the heart, liver, and kidneys show parenchymatous degeneration. The mucous membrane of the mouth is congested and swollen, chiefly at the base of the tongue ; the tonsils are enlarged and inflammatorily infiltrated ; and the salivary glands are hyperæmic. The mucous membrane of the pharynx and larynx is reddened, swollen and even studded with small hæmorrhages. Various kinds of foreign bodies are sometimes found in the pharynx and œsophagus. The stomach frequently contains straw, hair, feathers, pebbles, pieces of brick, particles of wood, bits of leather, portions of whipcord, and other foreign and indigestible substances ; but little or no food. Wortley Axe found foreign substances in the stomach of 90 per cent. of 200 rabid dogs which he examined *post mortem* ; but no trace of food. He considers that this condition of the stomach is the most important feature of the whole autopsy. Galtier found foreign bodies in the stomach in from 50 to 70 per cent. of 300 rabid dogs of which he made autopsies. The mucous membrane of the stomach is congested and swollen ; and small hæmorrhages and hæmorrhagic erosions are frequently seen on the surface of its folds. The intestine is generally empty, and its mucous membrane and the mesenteric glands are congested and swollen. The spleen is hyperæmic, swollen, and sometimes infiltrated with hæmorrhagic tumours. We find in the urine, albumen and sugar ; and in the case of dogs, biliary pigments. The respiratory mucous membrane is purple and the lungs are full of blood. The changes which are found in the brain and the spinal medulla are very inconstant. Sometimes we find hyperæmia, œdema and extremely minute hæmorrhages ; but at other times no changes are apparent. Csokor and others state that a considerable accumulation of lymph corpuscles may often be seen with the microscope along the capillaries and within the walls of the vessels and in the grey matter. The vessels of the grey matter are dilated and show microscopically small hæmorrhages and hyaline thrombi. According to Babes, some of the nerve-cells possess vacuoles and immigrated round cells, and manifest nuclear division ; and

the leucocytes of the dilated vessels contain characteristic, round, amœboid bodies which are pigmented and motile.

**Prognosis.**—Pasteur states that he has observed in his inoculation experiments with dogs, a few rare cases of recovery after the commencement of the first symptoms of the disease; but only when the attack was slight. Trasbot has seen 3 recoveries in rabid dogs, and Janson 1. There are no other absolutely reliable records of recoveries from rabies, which must therefore be regarded as an almost invariably fatal disease.

Hertwig found that only 37 per cent. of the animals inoculated by him became infected; and Renault 67 per cent. The comparative smallness of these figures must be due to the fact that in inoculations, the infective matter frequently does not adhere to the inoculation wound; that it may remain ineffective on the hair or on the surface of the skin; or that it may be washed away by the hæmorrhage caused by the inoculation. In the same way we may account for the fact that the bite of a rabid animal does not invariably produce rabies. It seems that on an average only from 20 to 30 per cent. of those bitten by rabid animals become infected. Hertwig puts the percentage at 5; Haubner, at 40; the Veterinary College of Lyons, at 20; and that of Alfort, at 33. Röhl calculated that in Austria during the years 1877–1887, the percentage among horses was about 40; among cattle and sheep, 50; among pigs, 36; and among goats, 20.

The percentage of deaths in mankind is considerably influenced by treatment. Bollinger states that of 100 bitten men only from 8 to 47 become infected. Pasteur puts the percentage at from 16 to 80.

**Therapeutics of Rabies.**—Treatment is purely prophylactic; for it is of no avail after the appearance of the symptoms of the disease. Although in men, we should always cauterise the wound with the hot iron, caustic potash, sulphuric acid, corrosive sublimate, etc., or excise the bitten part or the cicatrix; we should, for several reasons, apply such means to our domestic animals only in exceptional cases. Prophylaxis consists chiefly in the application of indirect protective measures, of which the best are those of the German police regulations of 23rd June, 1880, with regard to epizootics. The utility of this law was greatly enhanced by the introduction

of a tax on dogs, by which the number of dogs and consequently the number of cases of rabies were decreased, as was statistically proved in Bavaria. In large towns the compulsory wearing of muzzles, combined with preventive treatment of the wounds, and the establishment of a tax on dogs, have proved a most excellent means for checking the spread of rabies, provided of course that the muzzles effectively answer their purpose. In Berlin, for instance, the compulsory wearing of muzzles has greatly diminished the prevalence of rabies. The following statistics show the benefits to be obtained from these prophylactic measures. In Bavaria (tax on dogs, 1876; law in regard to epizootics, 1880) the number of rabid dogs decreased from 821 in 1873, to 11 in 1885; in Baden, from 53 in 1874 to *nil* in 1885; in Saxony, from 287 in 1866 to 16 in 1885; and in Prussia, where only the law for regulation of the epizootic was in force, from 672 in 1878 to 352 in 1885.

[In Great Britain, the regulations against rabies, which were first put in force in 1886, have proved singularly ineffective, considering that this disease is communicated only by the bite of rabid animals. The statistics of canine cases are as follows: In the year 1887 (217 cases); 1888 (160); 1889 (312); 1890 (129); 1891 (79); 1892 (38); 1893 (93); 1894 (248); 1895 (672); 1896 (438); and in 1897 (248). The faulty regulation of rabies in Ireland has long been a serious danger to life and property in Great Britain as well as in Ireland. The good effect of the muzzling order of 1896, despite its partial application, is shown by the following table of canine rabid cases:—

Month.	London. (Muzzling order put into force 7th Feb.)	Surrey. (Muzzling order put into force the whole year.)	Middlesex. (Muzzling order put into force 17th Feb.)
January . . . .	20	15	11
February . . . .	25	12	9
March . . . . .	22	11	4
April . . . . .	11	7	5
May . . . . .	11	3	5
June . . . . .	12	3	3
July . . . . .	14	4	3
August . . . . .	2	2	1
September . . . .	1	2	1
October . . . . .	4	2	2
November . . . .	2	1	2
December . . . .	4	2	2

As suggested by Hunting, the muzzling act, to be reasonably effective, should be put in force—immediately a case of canine rabies is reported—throughout the surrounding country, say, for a radius of 10 to 20

miles ; and should be continued in that area for a term sufficient to cover a fairly long period of incubation, say, for not less than 3 months. During an outbreak, every wandering dog which has been taken up by the police, if not destroyed in the meantime, should be segregated in a separate cage for at least 8 days (see p. 368), so as to make certain that the wandering was not due to the effects of rabies. Until Ireland gets a clean bill of health as regards this disease, the importation of dogs from it into Great Britain should be allowed only under quarantine rules similar to those at present in operation with respect to foreign countries. Such importation, except in the case of practically rabies-free countries such as South Africa and Australia, remains a constant danger to the United Kingdom, and should consequently be permitted only under a quarantine of at least 3 months' duration. It should be remembered that rabies is particularly frequent in Russia. The registration of all dogs in the United Kingdom would be a very desirable enactment, especially, if at the same time a rigid scrutiny into claims for exemption from the dog-tax on the plea that the animals are required for herding purposes, was instituted. At present a large number of dogs escape this tax, solely because they belong to sheep-dog breeds (collie or bob-tailed) and are owned by country people ; although they are never employed with farm stock. For valuable suggestions by Hunting and others, regarding the prophylaxis of rabies, see *Veterinarian* for July, 1897.—Tr.]

### **Pasteur's Protective Inoculation against Rabies.**

—Pasteur stated in 1884 that the virus of rabies could be gradually weakened, by carrying it from dogs to monkeys and by repeated transmissions from monkeys to monkeys. He also pointed out that the virus thus weakened, when injected under the skin or under the dura mater, after trephining the skull, did not produce rabies in dogs, but rendered the inoculated animals immune both to natural and artificial infection. In more recent times, Pasteur published another process of immunising by the use of the dried spinal cord of a rabid animal as the inoculation material. The spinal cord and its membranes are removed from a rabid rabbit under aseptic precautions as far as the cerebellum, and cut into pieces of 6 cm. in length. These pieces are tied to threads, which are suspended in bottles ; and the bottles are filled up to a height of 1.5 cm. with caustic potash, and closed with cotton-wool plugs. The bottles are then kept at a constant temperature of 20° C. After 3 or 4 days the portions of spinal cord dry up to the form of friable bands, the virulence of which gradually decreases according to the duration of the desiccation. For instance, rabbits become affected in 7 days, after inoculation with material which has been dried for 24 to 48 hours ; in 8 days, when the desiccation has lasted from

3 to 5 days; and in 15 days when it has been continued for about a week. Animals may be made immune to rabies by at first inoculating them with very attenuated material, and then gradually increasing the strength, so that they can finally bear the strongest virus with impunity. These attenuated inoculations sometimes cause the inoculated animals to become affected with a temporary and partial rabies. According to Babes, it frequently happens that dogs temporarily exhibit, from 8 to 14 days after the inoculation, depression, loss of appetite, emaciation, nervous symptoms, morbid desire to bite, and even paresis of the posterior extremities.

Pasteur states that the effect of the virus increases and the period of incubation diminishes, in proportion to the number of rabbits and guinea-pigs through the bodies of which the disease is transmitted. The strongest virus is prepared as follows: the experimental rabbits having been trephined with a trephine of 6 mm. diameter, a small quantity of the central nervous system of a rabid dog that has just died is injected aseptically under the membranes of the brain with a Pravaz syringe which has its needle bent at a right angle; the result being that dumb rabies breaks out in 15 days. If the injection be made subcutaneously, the development will be slower. When, after this, the virus is transmitted through a series of rabbits, the period of incubation, as we have already said, becomes gradually shorter, namely, 8 days in the twenty-fifth rabbit, and 7 days in the fiftieth rabbit, with which the maximum of virulence is reached. With an inoculation of ordinary fresh virus, the period of incubation is 15 or 16 days.

These statements of Pasteur were tested by a French commission, among whom were those eminent savants—Paul Bert, Bouley, Villemin, Vulpian, and Tisserand. This commission fully confirmed Pasteur's statements, and consequently there is no doubt that the virus of rabies can be either weakened or strengthened. Also, we cannot deny the possibility, in the future, of rabies being successfully combated prophylactically by protective inoculation.

Pasteur attempted to immunise human beings after they had been bitten, by inoculating them with attenuated virus. He applied this process first to dogs with a positive result. Altogether there have been inoculated from 1886 to 1890 by Pasteur and in the 20 Pasteur institutions in Russia,

Hungary, Austria, Italy, etc., over 10,000 (in France alone over 9,000) bitten people in the following manner. At first a piece, 2 or 3 cm. long, of the dried spinal cord of a rabbit is rubbed down in sterile broth, and this fluid is injected directly under the skin of the abdomen in a quantity of from  $\frac{1}{2}$  to  $\frac{3}{4}$  c.c. To begin with, the weakest inoculation material is used and then the strength is daily increased until finally a material of the highest virulence is employed. It is stated that by this process the mortality in rabies has been reduced from 16 to 0.5-2 per cent. (Pasteur, Cantini, Metschnikoff, Ullmann, Bujwid, Bardach, and others). Von Frisch, Bordoni-Uffreduzzi, de Rentzi, Högyes and other authorities declare that their researches prove that no benefit can be derived from Pasteur's supposed curative method. Consequently it is impossible at present to form a definite conclusion on this subject.\* Even admitting the efficacy of Pasteur's method of curative inoculation, we must not overlook the following points: First of all we do not know how many of the bitten people were really infected; the percentage being, according to Bollinger, only from 8 to 47. Pasteur puts the minimum at 16 per cent. Also, we cannot tell, in many cases, whether the bite has been inflicted by a rabid animal or by one merely suspected of rabies. In spite of protective inoculations by, respectively, Pasteur, Ullmann and Metschnikoff, a certain number of the patients die. The slight mortality may be accounted for by the fact that in nearly all cases the wound has been previously cauterised. Pasteur's method of inoculation is in any case too crude to deserve general adoption. In some institutions, exclusive of that of Paris, the method is strikingly unsuccessful. It has happened that supposed improvements of the method (Mailand) have accelerated the attack of rabies, or even originated it in inoculated persons. From the foregoing considerations we must agree with Bollinger that the establishment of Pasteur institutions is premature, at least, in Germany. The protective inoculation of dogs is considered even in France to be absolutely impracticable (Nocard). Statistics prove that Government regulations for the contagious disease of animals, tax on dogs, and compulsory wearing of muzzles in large towns, are amply sufficient to successfully combat rabies.

\* The large majority of English doctors and veterinary surgeons are strongly of opinion that the application of Pasteur's method of protective inoculation to persons bitten by rabid animals, greatly diminishes the risk of the disease becoming developed.—Tr.



**More recent Methods of Protective and Curative Inoculation.**—1. Högyes uses inoculation material in different strengths. He prepares these concentrations from the spinal cord of rabid rabbits with salt water (1 to 5,000, 1 to 2,000, 1 to 500, 1 to 200, 1 to 100, and 1 to 10) and makes successive inoculations, beginning with the weakest concentration. By this method dogs can be rendered immune against both the natural virus of rabies and the fixed virus. Högyes' experiments seem to prove that immunity may be conferred by diluted fresh virus repeatedly inoculated a short time either before or after the animal has been bitten. In 13 cases of dogs which were inoculated subsequent to infection, recovery took place after pronounced symptoms of rabies had appeared. In man, however, recovery from rabies by means of such inoculations has never taken place after the appearance of the symptoms.

2. Helmann and Ferran y Clua have shown that the greater the quantity of Pasteur's inoculation material (brain emulsion) that is injected, the more rapidly and the more certainly is immunity obtained. 30 to 60 c.c. of the emulsion have, for instance, been injected subcutaneously in one dose. The tolerance of the body to such large doses depends on the fact that the virus is injected only under the skin. Should other parts, such as muscles and nerves, be injured at the same time, the inoculation may be followed by an attack of rabies.

3. Babes states that Pasteur's method, though effective for obtaining immunity after the bite of rabid dogs, does not succeed in the case of bites by rabid wolves, for which he uses the blood of immunised dogs, and injects 20 grammes of it for each dose in the abdominal region of the bitten person. This inoculation of blood is stated to have been successful in 24 cases.

4. Galtier has demonstrated that herbivora and omnivora may be rendered immune by intravenous injections, not only against the consequences of subsequent bites, but also against bites already received.

5. Protopopoff placed pieces of virulent spinal cord for, respectively, 30, 46, and 60 days in glycerine broth, and, having thus deprived it of its virulence, he found that one or two intravenous or subcutaneous inoculations conferred immunity against the infection of rabies, and also prevented the appearance of the disease after infection. Out of 19 dogs which had thus been protected, 10 remained healthy after having been infected with the strongest rabic virus, even after it was injected into the brain. Protopopoff actually succeeded, by his method, in preventing an attack of rabies after intracranial infection. Of all the methods of anti-rabic protective and curative inoculation, his procedure seems to hold out the greatest promise for the future.

#### CANINE RABIES.

**Symptoms.**—The clinical appearance of rabies in dogs as in other domestic animals, occurs in two forms, namely, furious madness, and dumb madness; the former being the more frequent. Pasteur considers that furious madness takes place when the brain is attacked, and also when the virus has been directly inoculated into the brain; and that dumb mad-

ness is manifested when the spinal cord is specially invaded, and when the inoculation material has been applied subcutaneously. The two are consequently only different forms of one and the same disease, and are not different diseases, as was held in former times. A furiously rabid dog can transmit dumb madness, and *vice versâ*. We must also lay stress upon the fact that there are many intermediate forms between these two varieties, which often merge into each other so intimately that a distinction is impossible.

Rabies runs a highly typical, acute, and absolutely fatal course.

1. *Furious rabies* has three stages :—

- (a) The premonitory or melancholy stage.
- (b) The irritative or maniacal stage.
- (c) The paralytic or final stage.

(a) The *premonitory stage* of rabies lasts on an average from 12 to 48 hours; and sometimes even longer. The preliminary signs consist especially in the altered behaviour of the animal, which becomes capricious, sullen, peevish, excited, nervous, irritable, frightened, restless, distrustful, or obstinate.\* It likes to hide itself, frequently changes its resting-place, and often starts up suddenly; or it may become extraordinarily affectionate and confiding. Some dogs suffer from an abnormal itching of the site of the bite, which they gnaw and lick. There is a characteristic perversion of taste which has much similarity with that in pica ("licking disease") of cattle. Although at the commencement of the disease the patient has a good and sometimes a voracious appetite, later on it turns away from its accustomed and even favourite food. The affected dog is fond of licking cold objects; bites and gnaws at whatever comes across its way, and sometimes swallows foreign bodies, especially straw, grass, earth, stones, pieces of wood, bits of glass, rags, and even its own fæces and urine. Some dogs continually smell or lick their sexual organs or those of other dogs. In this stage we may sometimes notice slight spasms in swallowing, inclination to vomit, symptoms of choking, vomiting, coughing, panting, difficulty in breathing, fever, and continued stretching out of the neck. The patient generally suffers from constipation.

(b) The *stage of irritation* lasts 3 or 4 days, and is

\* The altered behaviour often takes the form of increased vivacity and increased friendliness of disposition. This high-strung, demonstrative state lasts only about a day or so, and then rapidly changes into the melancholy stage.—TR

characterised by attacks of fury, which may continue for some hours, and convulsions with remissions. Among other signs of restlessness, the dog evinces a violent desire to run away from its home. With this object, very quiet and domesticated dogs, which have been kept in confinement, destroy their kennel or break their chain; and dogs that live in the house remain close to the door so as to escape; and when they get into the open they wander about aimlessly, run to and fro, depart on long journeys, or boldly enter strange houses or yards. They often cover in a very short time great distances, 30 or 40 miles a day for instance. In some cases they return home. At the same time they are seized by a more or less excessive morbid desire to bite, which may increase to senseless fury and true mania. At first this inclination to bite is only slight, and is evinced by the dog snapping at inanimate objects, animals or people, and also, in its state of mental confusion, at imaginary objects in the air (snapping at flies). It is also very irritable. Later on it bites, sometimes blindly, sometimes aggressively, everything that comes in its way, such as human beings, including its own master (especially if he provokes it by punishment), horses, sheep, goats, fowls, and particularly dogs and cows. It will even bite iron bars that are held out to it, and live coals. Under such circumstances it frequently bites with such force that it breaks its teeth, or is unable to unlock its jaws. Some dogs direct their fury against themselves, and lacerate their tails, genitals, and legs even to the bone. In rarer cases they carefully avoid man and beast. In a few exceptional instances they show no desire to bite human beings. In this stage we must attach great diagnostic importance to the alteration of the voice, which manifests itself as a peculiarly hoarse, rough, howling bark, the first notes of which are prolonged into a high-pitched, long-drawn-out howl. Paralysis of the vocal cords is the probable cause of this change in the voice.

In other canine patients, depression is more prominent than maniacal excitement. They appear dull and imbecile, have hallucinations, stare constantly at one spot, snap at imaginary flies, howl continually, and are perfectly insensible to blows and kicks. In a few cases well-trained dogs obey the commands of their master to the very last.

(c) In the *paralytic or final stage* the animals are usually very much emaciated, frequently to a skeleton; they become disfigured, and often quite unrecognisable; the hair stands

on end; the eyes are deeply sunken, staring and glassy; and the appearance is loathsome and weird. At this stage, various forms of paralysis appear; first of all, that of deglutition, in consequence of which the animal can neither swallow nor excrete saliva. Next comes paralysis of the lower jaw, which drops down; the mouth remains open, and the tongue hangs out. This condition is followed by paralysis of the hind-quarters, which manifests itself in staggering, stumbling, and by increasing bodily weakness. The muscles of the tail, rectum, and urinary bladder become paralysed, with symptoms of weakness and depression, which are varied by paroxysms of excitement that gradually become rarer and rarer. Finally the animal dies between the fifth and eighth, or, as a maximum, on the tenth day of the disease, from paralysis of the brain and general exhaustion. Paralysis of the hind-quarters sometimes appears in the early stage of the disease, before the morbid desire to bite sets in (Zschokke).

Only a few observations on the temperature during rabies have been made. According to Hertwig, the temperature rises about  $3^{\circ}$  C. or more, and falls rapidly towards the end—a statement which we can confirm from our own experience in one case. Müller found in 2 cases a temperature of  $40^{\circ}$  C. 24 hours before death.

2. *Dumb madness* is distinguished from the furious form chiefly by the absence or very short duration of the stage of irritation or mania. Consequently paralysis, especially of the lower jaw, appears comparatively early. Death takes place in 2 or 3 days.

Several observers state that they have seen recovery after the first stages of rabies. Such instances are certainly extraordinarily rare.

We may see from the foregoing description of the course of rabies in dogs, that the views current among laymen with respect to the symptoms of this disease in these animals are very erroneous. According to popular opinion, rabid dogs always run straight ahead with their tail between their legs; they show great aversion from water; their eyes are excessively red; saliva and froth flow from their mouth, etc. The fact, however, is that rabid dogs not only have no dread of water, but they frequently lap it, and will even swim in rivers and lakes.

**Diagnosis and Differential Diagnosis.**—Positive proof of a bite having been inflicted is of primary importance in the diagnosis of rabies in dogs. The chief clinical symptoms are: changed behaviour, altered voice, desire to run away,

aggressive and morbid inclination to bite, paralysis, especially that of the inferior maxilla, and the typical course of the disease. If really rabid dogs are segregated and watched, the symptoms just mentioned will become apparent in a few days. If to this we add the negative result of the *post mortem* examination, we shall be able to make a pretty certain diagnosis of rabies. The presence of foreign bodies in the stomach is, in conjunction with other suspicious circumstances, a valuable diagnostic factor ; but it is not sufficient of itself to positively prove rabies, in which disease it sometimes happens that the stomach contains no foreign bodies. Besides, foreign bodies may be found in the stomachs of non-rabid dogs. Sometimes the knowledge of the place from whence the suspected dog came is of diagnostic value. We have, for instance, diagnosed rabies in a dog by the fact that the animal has been recently brought to Berlin from Russia, which is a highly-infected country. The difficulty of forming a correct opinion is greater in dumb madness, in cases of well-trained dogs being affected, and when the medical history is incomplete, than under opposite conditions. In the former cases, diagnosis can be made only by the intracranial transference of a fresh piece of brain or spinal cord to a dog, or rabbit, for instance. This method is especially applicable to cases in which human beings have been bitten. Instead of intracranial inoculation, which demands great attention to detail, Nocard recommends that after depriving the cornea of sensibility with cocaine, the anterior chamber of the eye of a healthy dog should be injected by means of a Pravaz syringe, with a piece of the medulla oblongata rubbed down with distilled water. By this procedure rabies appeared in from 14 to 17 days in over 200 instances. We may perform the inoculation with greater precision by carefully rubbing down with a little distilled water a small portion of the bulbus of the medulla oblongata, straining the fluid through fine linen, and taking up the filtrate in a Pravaz syringe. Ten to 15 drops of a 5 per cent. solution of cocaine are dropped into the space between the lower eyelid and eye-ball of a dog which is firmly secured. When the cornea has lost its sensibility, it is perforated with the needle of the syringe and 1 or 2 drops of the emulsion are injected into the anterior chamber of the eye. The emulsion should be made as aseptic as possible, so that the eye may not suppurate. Intracranial and intra-ocular injection generally produces furious madness ; sub-cutaneous and intravenous inoculation, dumb madness.

There are many diseases which may be confused with rabies, such as hyperæmia of the brain, inflammation of the brain, parasites and tumours in the brain, distemper, epilepsy, inflammation of the stomach, intestinal inflammation, intestinal parasites (especially *tænia echinococcus*), foreign bodies in the stomach and intestines, rupture of the stomach (Poisson), perforation of the intestinal canal by *tænia serrata* (Cadéac, Wolpert, and Lahogue), parasites in the nasal cavities (*Pentastomum tænioides*), pharyngitis, bones wedged between the teeth (Johne), foreign bodies and parasites (*tænia*, *spiroptera sanguinolenta*) in the pharynx and œsophagus, paralysis of the maxilla in consequence of an affection of the *nervus trigeminus* (Vermast, Munich, and Cadéac), paralysis of the facialis, hemiplegia (Sewell), great excitement caused by removal of the puppies from the bitch (Colin), sunstroke (Fünfstück), love of biting, bad temper, *eustrongylus gigas* in the kidneys, etc. In all doubtful cases inoculation is the best test.

**The symptoms of rabies in man** are not quite the same as those in dogs, although the disease in both runs through the three typical stages which we have described. In the premonitory stage there is general malaise, pains in the cicatrised wound which was caused by the bite, swelling of the neighbouring lymph glands, and aversion from fluids. In the second stage, reflex spasms, delirium, and mania appear. The reflex spasms affect the muscles of deglutition, and those of the pharynx, and become excited particularly by the sight of water, by attempts at swallowing (*hydrophobia*), by the feeling of thirst, and even by the mere thought of drinking. These reflex spasms also affect the muscles of respiration and those of the general system. At the same time an excessive feeling of anxiety and uneasiness is present. Thirst is greatly increased, and the patient slavers on account of not being able to swallow the saliva. The temperature of the body is sometimes only slightly increased, but at other times it is considerably raised. The third or paralytic stage is distinguished by paralysis and spasms, and death takes place in from 2 to 4 days. It has been stated that a few recoveries have been observed. Treatment, apart from prophylactic cauterisation of the wound, consists in bringing on a general narcosis by chloroform, chloral hydrate, potassium bromide, and by the tentative administration of curare.

#### BOVINE RABIES.

**Etiology.**—Of all the domestic animals, next to dogs, cattle are most liable to become affected with rabies.\* Not

\* During the years 1887 to 1896, inclusive, the following were the respective numbers of animals which were reported to have been attacked by this disease in Great Britain: 2,386 dogs, 259 deer, 78 sheep, 43 cattle, 21 horses, and 20 swine. The high position in this list occupied by deer is due to the fact that 257 deer were attacked in one year (1887).—Tr.

only are cattle more numerous than horses ; but dogs are more aggressive to the former than to the latter. Rabies in ruminants is usually caused by the bite of dogs, and seldom by one ruminant biting another ruminant, as observed by Cope and Horsley among the red-deer in Richmond Park ; or by rabid horses, cats, or pigs. The site of the bite is generally on the fore part of the head (generally on the lips), and on the hind-quarters. The period of incubation averages from 4 to 8 weeks, and rarely extends to several months. Mieckley proved in 1 case that it lasted for 327 days ; and Morro records 1 of 2 years and 4 months. The minimum duration is 1 week. Spinola states that pregnancy in cows prolongs the period of incubation. As already mentioned, 2 cases of supposed hereditary transmission of rabies to the calf have been published.

**Symptoms.**—The furious form of rabies is more frequent in cattle than dumb madness. The characteristic symptoms generally manifest themselves by butting with the horns and stamping with the feet. The attacks are often so violent that the horns become broken. Other affected beasts dig up the ground with their horns and feet. The morbid desire to bite is less marked than in dogs. The expression of the face is generally wild ; the eyes staring and prominent ; and the conjunctiva congested. Sometimes the animal continually bellows with a peculiar alteration of the voice. Excessive secretion and dribbling of saliva, and continuous constipation with severe straining to defecate, are constant and striking symptoms of rabies in cattle. This tenesmus may be the first visible sign of the disease. Some patients yawn continually for hours. In many the sexual desire is increased. At a later stage the movements of the hind-quarters may become stiff and straddling, and general paralysis may set in. There is finally excessive emaciation, and death takes place in from 4 to 6 days. Ladague has observed, in a few cases, an intermittent course of rabies in cattle.

**Differential Diagnosis in Cattle.**—We have here to consider : inflammation of the brain, especially tubercular basilar meningitis ; poisoning, particularly lead-poisoning ; gastrointestinal inflammation (Papa) ; foreign bodies in the cesophagus ; new growths on the reticulum (Meyer) ; dermanyssus mites in the outer auditory passage (Stadler and Schuemacher) ; malignant catarrhal fever ; anthrax ; rinderpest ; sunstroke ;

excitement in consequence of deprivation of liberty (Köhne); etc.

#### EQUINE RABIES.

**Etiology.**—Rabies in horses is nearly always transmitted by the bite of rabid dogs; in rare instances, by that of wolves or foxes. The period of incubation generally lasts from 4 to 8 weeks, with a maximum of 12 weeks, which, however, appears to have been exceeded in a few cases; for Röll observed one of 283 days; Zündel, one of 378 days; and Gotteswinter, one even of 20 months.

**Symptoms.**—Rabid horses at first exhibit unsteadiness, restlessness, and terror. They paw the ground with their feet, neigh, gnaw and bite at the manger, partitions and articles of stable gear; and sometimes manifest an itching irritation on the site of the bite, which is mostly found on the lips, muzzle, or forelegs. Some, but by no means all, rabid horses are aggressive to mankind and become seized with an irresistible impulse to bite and kick. Some lacerate and mutilate their own bodies by tearing the skin, testicles, etc. Others will rush like a dog at a stick if it is held out to them, and will bite and pull at it. By the violence with which they bite hard objects, they sometimes break their incisor teeth, and even the body of the inferior maxilla. Certain horses remain in a dazed condition; and though at first they eat ravenously, they will not touch their food later on. Other horses masticate everything that gets between their teeth: they even take up their dung and swallow it, and evince severe thirst. Great desire to micturate and increased sexual excitement are characteristic of equine rabies. Stallions try to mount mares, have erections, and involuntarily eject semen. Mares become violently in season, scream, strain to urinate, and manifest excessive irritation of the pudenda. When paralysis of the muscles of deglutition sets in, the food is returned through the nose; the animal works its jaws continually, as in chewing, grinds its teeth, shakes its head, and swishes its tail. These symptoms are associated with convulsions and muscular spasms in different parts of the body, especially in the muscles of the head (lips and cheeks), neck, abdomen, and thorax. Symptoms of colic, such as groaning, frequent lying down and getting up, straining to defecate and even prolapse of the rectum may occur. Finally, general paralysis sets in, the animal staggers in its walk, knuckles over, gives way at the fetlocks,



and exhibits a groping uncertain gait, and, as a rule, soon becomes paralysed in the loins. In some cases, paralysis appears at first in the muscles adjoining the wound, as on the lips or on a forefoot (Gerlach and Mégnin). The temperature of the body rises from normal to 40° C. or more; and the beats of the pulse may be increased to double or treble their usual number. Towards the end there is frequently a general outbreak of sweat. Death often takes place by apoplexy as early as 24 hours. The average duration of the disease is from 4 to 6 days. Dumb madness, in which the morbid desire to bite is absent, is more common in horses than in cattle.

**Differential Diagnosis.**—In horses the following diseases may be mistaken for rabies: Inflammation of the brain, staggers, abscesses of the brain (Kopp), excessive horsing, colic, inflammation of the stomach (Olivero and Allemanno), rupture of the stomach, and paralysis of the loins. The psychological disturbances, morbid desire to gnaw and bite, straining to defecate and micturate, symptoms of colic, local itching irritation, and increased sexual desire are phenomena of diagnostic importance.

#### RABIES IN CATS.

**Symptoms.**—After an average period of incubation of from 2 to 4 weeks, rabid cats show great inclination to run away from home, and a strong desire to bite and scratch. The injuries caused by rabid cats are more dangerous than those inflicted by any other domestic animals; because cats jump up on persons, and try to lacerate and scratch the face. They also attack dogs, of which they show no fear. Their voice becomes characteristically altered and hoarse. Death takes place between the second and fourth day. Rabies of cats may be confused with helminthiasis. Next to dogs, cats are the most frequent agents by which rabies is transmitted to man. Pasteur estimated that among 12,000 persons who had been bitten by rabid animals, 11,000 were bitten by dogs, and more than 700 by cats.

#### RABIES IN PIGS.

**Symptoms.**—Rabid pigs show at first great restlessness, run wildly about, scream, squeak, dig up their litter, hide themselves, attack man and beast, and exhibit a great desire to bite. They secrete an abnormally large amount of saliva, slaver, and frequently swallow foreign bodies. At the commencement of

the outbreak, the infection wounds often manifest a well-marked inflammation, which induces the animals to rub and gnaw the wounds. It has been stated that dumb rabies has not been observed in pigs. The duration of the disease is very short, and usually lasts only 1 or 2 days; frequently only 1 day. The period of incubation is on an average 2 or 3 weeks. According to the Prussian statistics of epizootics, it varies from 6 to 179 days.

#### RABIES OF SHEEP AND GOATS.

**Symptoms.**—Rabid *sheep* show great restlessness, and considerable pruritus at the site of the bite, which they lick and gnaw, and pull out the wool near the part. Sexual desire is usually considerably increased. The affected animals crowd and push against their fellows, try to jump on them, bleat with a dull and rough voice, and lose all shyness and timidity towards mankind. If people approach them they become very restless, stamp with their feet, bite at a stick if it is held in front of them, and will bite even stone. They try to jump up on walls, snort wildly, and assume a glaring and staring expression of face. Excessive weakness with paralysis of the hind-quarters soon sets in, and the sufferers generally die in from 3 to 6 days. The period of incubation is on an average 3 or 4 weeks. In most cases, the infection takes place through dogs, by which a great part of an entire herd may become rabid. In rare cases the infection is due to a rabid wolf or bull (Bourrel!).

The symptoms in *goats* are essentially the same as in sheep.

#### RABIES OF BIRDS.

**Symptoms.**—Rabid birds are very easily frightened. They become extremely restless, run about continually, screech, and jump about in a disordered manner. They attack each other and even human beings with beak and claws, and may thus tear pieces of cloth from people's clothes and try to swallow these bits of cloth. Their voice soon becomes hoarse; they stagger; become paralysed; and die in 2 or 3 days. The period of incubation is about 6 weeks, with a maximum, it is stated, of 11 months.

## FOOT AND MOUTH DISEASE.

Foot and mouth disease in cattle—Foot and mouth disease in sheep—Foot and mouth disease in pigs—Infectious aphthæ of horses—Infectious aphthæ of dogs—Infectious aphthæ of cats and rabbits—Infectious aphthæ of birds—Transmission of mouth disease from cattle to man.

**History.**—Foot and mouth disease (*aphthæ epizooticæ*) has been known for a very long time. It was well described by some observers in the middle of the eighteenth century, when it broke out with great violence, especially in Germany and France, It raged from 1809 to 1812 and from 1819 to 1823, particularly in Southern Germany, Switzerland, and Italy (Buniva, Handel, Lux, and others). In 1839 it was introduced into England, where, since then, ten great outbreaks have taken place (Brown). In the forties (1845–1846) and fifties (1855–1857) it spread over the whole of Europe. Among recent epizootics we may mention those which occurred in the years 1862, 1869, 1871–1874, 1875–1877, and 1883–1884. During 1871, 700,000 head of cattle, both in England and in France, became infected, with a loss of 7,000 head (1 per cent.); during 1872 in Württemberg alone, 50,000 head, with a mortality of 1,500 (3 per cent.); and during the same period in Baden, 150,000 head of cattle. During 1883 in Great Britain, the disease attacked nearly half a million; in Prussia, Austria, and Italy, 60,000 in each country; and in Bavaria, over 100,000 animals (83,000 cattle, 15,000 sheep and 5,000 pigs). In the German empire during 1890, over 800,000 animals became infected; and during 1892 over 4,000,000 (in Prussia alone over 3,000,000). The last great epizootic invasion of foot and mouth disease in Germany took place during the years 1890 to 1892. In some years this usually benign disease has assumed a very malignant type, particularly in 1892. For instance, the loss in Bavaria alone during the months of August, September and October of that year was over 3,000 head of cattle. Similar experiences have been made in former times. Thus, 2,000 head of cattle died from foot and mouth disease during 1839 in the cantons of Bern and Freiburg. During 1872 there were heavy losses from it in France and Bavaria.

**Etiology.**—Foot and mouth disease is an acute infective disease belonging to the group of acute exanthemata. Its contagium is both fixed and volatile and can be transmitted either

directly or indirectly by intermediate bearers to other animals. The infective matter, the nature of which is not fully known, is contained in the fluid of the vesicles, discharge of the ulcers, saliva, milk, fæces, urine, expired air, perspiration, and exhalations from the body.

The vitality of the contagium varies greatly, according as it is in a moist or dry condition. It quickly becomes ineffective by desiccation. Cattle which have been attacked lose the power of transmitting the disease shortly after the lesions on the mouth, feet, and udder have healed; and pigs, as soon as the vesicles on the feet have dried up, and the excoriated parts of the skin on the coronet and in the cleft of the hoof have become covered with dry, firmly-adhering scabs. In cows, the excretion of infective matter ceases with the healing of the morbid process. The dung which accumulates between the toes loses its contagiousness, as a maximum, within 14 days, which is also the extreme limit for the virus to remain effective in the fleeces of diseased sheep. The contagium continues active for a longer time in dung and in damp substances. It is destroyed by strong disinfectants and by boiling, as in milk.

The disease spreads as an epizootic along the line of high roads, and frequently from east to west, in a very short time over large tracts of country, as for instance, from the Caspian Sea to the Atlantic Ocean. The more extensive is the network of railways in a country, the more rapidly does the disease become disseminated through it. The chief agents by which the infection is spread are cattle-trucks, stalls in inns, cattle-markets, cattle-dealers, veterinary surgeons, drovers, common pastures, common drinking-troughs, herds of sheep, bulls, infected fodder, litter, manure, skimmed milk, and the waste products of dairies, especially of large amalgamated dairies. Herds of swine that are driven along high roads are particularly dangerous from an infective point of view. In sheds, direct infection takes place by the animals licking each other and by the inspiration of contaminated air. The disease may be transmitted indirectly by attendants, especially when milking. Sucklings are infected directly by the milk. An intra-uterine infection may also take place.

Nothing certain is known with regard to the ports of entrance of the contagium into the body. It is probable that the virus can be absorbed by the digestive apparatus as well as by the lungs. Any species of animal may infect any other; for instance, cattle and sheep can be infected by pigs; sheep, by

cattle, and *vice versa*. The disease is sometimes spread by infected red-deer and fallow-deer.

**Bacteriology.**—Nosotti states that he has constantly found in the pure contents of the vesicles, a micrococcus which can be easily stained in a solution of aniline. He made pure cultures of it in nutrient material outside the body, and inoculated it successfully. Klein claims that the infective matter of foot and mouth disease is a micrococcus which he cultivated artificially, and which produces characteristic cultures as a diplococcus and streptococcus, 20 or more cocci being sometimes attached to one another. Klein made these cultures on suitable nutrient material (solid blood serum and solid nutrient gelatine), and found that they assumed the appearance of soft, punctiform, granulated membranes, which consisted of small drops lying close together. He states that he produced foot and mouth disease in sheep by feeding experiments with these cultures, but that subcutaneous inoculations gave negative results. Schottelius states that the cause of infection is spherical formations which are attached to each other in the form of a rosary, and which resemble streptococci, and consequently he has given them the name of *streptocytes*. Subcutaneous injections of pure cultivations produced in cattle a feverish affection accompanied with salivation. Kurth found a similar organism, and called it *streptococcus involutus*, the cultures of which gave negative results by inoculation.

**Occurrence.**—Foot and mouth disease is a malady peculiar to ungulates, and therefore occurs chiefly in cattle, sheep, pigs and goats. Horses, dogs, cats, and birds are less frequently attacked. Human beings become easily infected. All wild even-toed animals (red-deer, roebucks, fallow-deer and chamois) and wild ruminants in zoological gardens (camels, llamas, giraffes, antelopes, aurochs, buffaloes, yaks, zebu,\* etc.) are liable to it. It is stated that draught-oxen possess less predisposition to it than other cattle (Peschel). An attack confers no subsequent immunity. In fact, an animal may suffer several times from this malady in the course of a year.

Foot and mouth disease is of great importance from an agricultural point of view; for the pecuniary loss is generally very severe, in consequence of the rapid manner in which this disease spreads through the entire stock of a country. From 25 to 50 per cent. of all the cattle may become infected; traffic and commerce suffer great restrictions in consequence of the legal enactments that are put into force; the loss in milk and its products is heavy; sometimes the mortality of the infected animals is comparatively high; and the value of the survivors is depreciated by decrease in their working powers, and by diminution

\* The zebu is not found in a wild state.—Tr.

in their bodily weight. The losses thus caused in 1883 in England were estimated at a million sterling; in France in 1871, at £1,500,000; and in Switzerland, at £400,000 yearly. The losses in Germany during the last epizootic were reckoned at £5,000,000.

**Statistics.**—In Germany, from 1886 to 1888, only 100,000 animals were infected with foot and mouth disease; but from 1889 to 1894, over 7,000,000 animals were attacked. The respective figures are as follows :—

Year.	Total.	Cattle.	Sheep and Goats.	Pigs.
1886	13,603	5,366	6,368	1,839
1887 . .	31,868	12,723	14,400	4,745
1888 . .	82,834	37,164	19,786	25,884
1889 . .	555,178	262,375	238,399	54,404
1890 . .	816,911	432,235	230,868	153,808
1891 . .	821,130	394,640	244,282	182,208
1892 . .	4,153,537	1,504,299	2,210,986	438,252
1893 . .	500,342	204,832	220,402	75,108
1894 . .	192,613	93,921	66,287	32,405
Total . .	7,168,016	2,947,555	3,251,808	968,653

The disease was absent from the German Empire in the second quarter of 1887, but broke out in the following quarter and continued to fluctuate in extent, until there were over 4,000,000 cases of it in 1892, from which time it decreased. The following factors favour its dissemination: introduction from neighbouring states; neglect or defective execution of the police regulations with regard to the importation of cattle; defective disinfection of sheds and cattle-trucks; unrestrained use of animals for draught purposes; unrestrained use of common pastures and of wells for drinking; cattle dealing; driving cattle along high roads; removal of manure; intermediate bearers, such as human beings and their clothes; cattle food; utensils; undressed skins; blood; horses; cats; birds; rats; flies; unsanitary dwellings; over-flowing of middens; and dairy companies. The skimmed milk and waste products which are returned by such dairies, and are given to pigs and calves as food, largely contributed to the spread of the disease. Stress was also laid upon the fact that the permitted practice of sending out the unboiled milk from healthy cows which were living in infected sheds was a concealed source of considerable danger. Favourable results in extinguishing the disease, and in restricting its dissemination, were obtained in several places by temporarily prohibiting the holding of cattle-markets, the suppression of which was not, as a rule, followed by any economic disadvantages.

[From 1886 to 1896, inclusive, the number of animals attacked by foot and mouth disease in Great Britain, were as follows: cattle, 1,295;

sheep, 3,673; and swine, 107. No animals were reported to have been attacked during the years 1887, 1888, 1889, 1890, 1891, 1895, 1896 and 1897. Since the year 1877, more sheep appeared to have suffered than cattle.—Tr.]

**Symptoms in Cattle.**—Foot and mouth disease manifests itself in cattle by the appearance of vesicles and ulcers on the oral mucous membrane (mouth disease), and on the skin of the coronet and on the skin of the interdigital space (foot disease). Sheep, goats, and pigs are usually affected only on the feet.

*Symptoms of the oral form of the disease in cattle.*—After a period of incubation of, on an average, from 3 to 5 days (with a minimum of from 36 to 48 hours), there is a moderate rise of temperature (up to 40° C.), which becomes normal as soon as the eruption appears. Appetite and secretion of milk become diminished; rumination is suppressed; the mouth is generally kept closed; and slight salivation takes place. After 2 or 3 days yellowish white vesicles, the size of a hemp seed, appear on the gums, especially on the toothless part of the anterior maxilla; on the edges, base and lower surface of the tongue; on the buccal mucous membrane; and on the lips. These vesicles gradually increase in size, so that they sometimes become as large as a five-shilling piece or even larger; and they also often become confluent. These vesicles, large and small, stand out prominently from the surface and contain at first a yellowish, limpid fluid, which gradually becomes turbid. The bursting of the vesicles leaves on the affected mucous membranes moist, highly reddened, and very painful erosions, denuded of epithelium. These spots become slowly covered with fresh epithelium or turn into ulcers with a foul base and finally cicatrise. At this stage of the disease there is a considerable amount of salivation and slaverling. The saliva continually runs in long threads out of the mouth and accumulates in the feeding-trough. The "smacking" noises thus produced by the movements of the jaws are very characteristic. The exanthema usually spreads to the muzzle. During the course of the disease the animals become greatly emaciated, with a loss of weight which may be as much as 1 cwt. The milk becomes altered and assumes a mucous, colostrum-like condition, a yellowish-white colour, and has a bad taste. It coagulates easily, and can only with difficulty be turned into butter and cheese. The quantity of the milk becomes considerably decreased, not only during the disease, but also for a comparatively long time after

recovery. This reduction varies, on an average, from 50 to 75 per cent. The exanthema in milch cows often spreads by the act of milking to the udder and teats.

The foregoing symptoms are those of the simple and ordinary form of the disease, which runs its course in from 8 to 14 days. At other times it assumes a malignant type which is entirely different from that of the benign form, and has been carefully studied only within the last few years. The most striking feature of the severe variety of foot and mouth disease, which may have very unusual sequelæ, is a fatal complication resembling apoplexy, at the height of the fever or during the period of convalescence, after the exanthema has healed. The animal begins to bellow and falls down dead quite suddenly, as if struck down by lightning, or dies in a few hours under symptoms of violent dyspnœa, discharge of blood from the nose, and great prostration. The cause of these apoplectic cases of death, which remind one of anthrax, is paralysis of the heart or paralysis of the lungs, produced by the toxins in the infective matter of the disease. This paralysis is somewhat similar to that which occurs in cases of chloroform-poisoning. There are also peculiar local manifestations of the affection in different parts of the body. The inflammation spreads from the canals of the teats to the parenchyma of the mammary glands and leads to the development of violent inflammation of the udder, followed by a sero-sanious discharge from the teats, induration, and atrophy. If the aphthæ appear on the pharyngeal mucous membrane, the animal will suffer from pharyngitis, difficulty in swallowing, regurgitation, and cough, and not uncommonly becomes affected with pneumonia due to foreign bodies, when drenches have been administered. Nasal catarrh and bronchial catarrh are often present, as in other acute exanthemata. If the exudative process in the oral cavity be very intense, croupy or caseous plate-like formations may appear upon the oral mucous membrane with disintegration of the cast-off epithelium, in consequence of which a fœtid odour may become developed in the mouth. Some affected animals, especially sucking calves, and also full-grown cattle, show in grave invasions of the disease, an intense gastro-intestinal affection with severe diarrhœa. We may sometimes observe inflammation of the matrix of the horn-cores, and vesicles on the skin at the base of the horns, which may even become shed. In a similar way we may find vesicles on the vulva, vaginal mucous membrane, entire skin, especially on the abdomen and chest, and even on the cornea. Abortion



is not infrequent during the attack and a short time after recovery. In a few instances the exanthema is entirely absent, in which case the manifestations of the disease somewhat closely resemble those of anthrax fever (subacute anthrax, p. 335). It occasionally happens that foot and mouth disease is complicated with pleuro-pneumonia, or other infective diseases.

*Symptoms of the pedal form of foot and mouth disease in cattle.*

—The pedal symptoms generally appear after the oral manifestations, although they may be the first to become developed. We observe first of all redness, abnormal heat and painful swelling of the skin at the coronet, and especially between the toes and towards the plantar cushions. These changes may appear on 1 foot or on all 4 feet at the same time. Minute vesicles the size of a pea up to a hazel-nut, spring up after a day or two on the inflamed skin, and soon burst. They are filled at first, as already stated, with a yellowish, limpid fluid, which later on becomes turbid and greasy. At the same time the animal becomes lame, moves stiffly and lies down a good deal. Under ordinary circumstances the vesicles heal completely in 8 to 14 days.

Also in the pedal form, grave complications are not rare. Severe inflammation may appear on the skin of the digits, especially when the disease has been neglected and when the animal has been compelled to stand on damp and soiled ground, or when it has had to travel over rough and stony roads. In such cases, the aphthous process becomes complicated with excitors of septic inflammation which lead, first of all, to an erysipelatous inflammation of the skin of the digits; and later on to the formation of ulcers and abscesses, panaritium in all its forms, purulent arthritis in the neighbouring articulations, necrosis of the bones, shedding of the hoofs, and finally to pyæmia. In such cases we find more or less considerable decubital gangrene.

**Differential Diagnosis.**—We must not confuse the ordinary mild form of the disease with injuries, cauterisations and scalds of the oral mucous membrane; aphthous and mycotic stomatitis produced by fungi in the fodder and known as “sporadic aphthæ;” actinomycosis of the tongue; stomatitis mercurialis; poisoning with potato-tops; variola; panaritium; inflammation of the cleft of the hoof; eczema of the back of the pasterns from feeding on distillers’ wash; and ordinary grease (eczema of the pastern). In one case Henninger saw an infection of the oral mucous membrane brought on by licking

greasy heels. The malignant variety of the disease is frequently confused with anthrax and sometimes with the bovine form of "deer and cattle disease" and rinderpest.

**Course and Prognosis.**—The symptoms of foot and mouth disease vary greatly in different years. They are sometimes slight; at other times severe. The course is generally benign and typical and almost always ends in 2 to 3 weeks (mortality from 0 to 1 per cent.). At other times the course of the disease is so malignant that there is a mortality of from 5 to 50 per cent. in full-grown animals and of from 50 to 80 per cent. in sucklings, which frequently die suddenly during the first days after birth. Weak and badly-fed animals are more liable to die from the disease than strong and well-nourished ones.

The duration of an outbreak in a shed or herd is from 4 to 6 weeks. Usually it spreads very rapidly although its dissemination may be slow at other times. As a rule the animals regain their condition very quickly after recovery. In exceptional cases, however, convalescence may be delayed by the sequelæ of the disease, such as emaciation, decreased milk supply, difficulty of breathing, inflammation of the udder, chronic affection of the hoof, pruritus, cutaneous eruptions with loss of hair, permanent lameness, etc. Besides loss of condition and decrease in the supply of milk, diminished fecundity also inflicts serious damage to owners.

**Anatomy.**—The changes found in malignant foot and mouth disease vary greatly. Death may be due to congestion and œdema of the lungs, and passive dilatation of the heart; to fatty degeneration, hæmorrhagic infarcts and cellular infiltration of the muscular portion of the heart; or to violent gastro-enteritis with intestinal hæmorrhage. Multiple embolic myocarditis has also been found to be present. The *post mortem* appearances are sometimes very slightly characteristic. We may find in the abomasum a violent inflammatory œdema of the mucous membrane with prominent gelatinous swellings; and in the first 3 stomachs, especially along the pillars of the rumen, aphthæ, ulcers, and hæmorrhagic foci. The mucous membrane of the small intestine is sometimes glassy, swollen, dark in colour, and infiltrated with inflammatory hæmorrhages, and the contents of the intestine bloody. Aphthæ have been found in the larynx and pharynx and on the inflamed tracheal and bronchial mucous membrane. In a few cases pleuritis,

hydrothorax, hydrocephalus internus and subcutaneous hæmorrhages were observed *post mortem*.

**Treatment.**—Prophylaxis consists in segregating the infected animals and in using the milk only after it has been well boiled. A purely dietetic treatment generally suffices in cases of the benign form of the exanthema; the animals being fed, if possible, on gruel. They should have a constant supply of pure drinking water and clean stalls. The floor should be kept free from moisture with dry litter, tan, gypsum or kainite. It is evident that medicines cannot cut short the course of the disease after it has commenced. Their action is essentially symptomatic and is needed only in severe cases. The ulcers on the mouth may be treated with disinfectants and astringent solutions (creolin, lysol, solutol, pyoktanin, alum, borax, tannic acid, and nitrate of silver); the foot disease with watery solutions of creolin, lysol, pyoktanin, tar, carbolic acid, bluestone, etc.; and the inflammation of the udder with ointments of creolin, lysol and camphor, and with salicylic or boric acid combined with glycerine.

**The Inoculation of Foot and Mouth Disease.**—As early as the beginning of last century (Buniva), and frequently since then, inoculations have been successfully applied for the purpose of shortening the progress of the epizootic in large herds and for inducing the disease to run a mild and uniform course (Ercolani, Brauell, Renner, Hoffman, Wirtz, Spinola, Hertwig, Lewes, Brandes, and others). Not only is the induced disease, as a rule, mild and confined to the mouth; but the practice of inoculation cuts short the duration of the epizootic, and, as a consequence, the duration of the protective police regulations. We are, of course, considering the mild form of the disease; for inoculation with the malignant variety would be too dangerous to put in practice. The inoculation is carried out by placing some of the saliva of an infected animal into the mouth of the beast which is to be inoculated, after slightly excoriating the mucous membrane; by inoculating it anywhere under the skin with a lancet; or by drawing impregnated threads through the skin, preferably through that of the ear and tail. Pigs may be inoculated with a vaccinating needle on the snout. A rise of temperature takes place 24 hours after the inoculation, and aphthæ appear on the third day, burst on the sixth day, and heal about the tenth. Such inoculations made during the last

few years in almost all parts of Germany have been generally attended with good results, especially when put into practice at the very beginning of an epizootic. On an average the inoculations "take" in from 50 to 80 per cent. of the animals; the remainder proving to be immune.

On the other hand, *protective inoculation* is not justified; because one attack of foot and mouth disease does not confer subsequent immunity. As we have already stated, animals may suffer from the disease more than once in the same year. In fact we may sometimes find that one attack follows another attack within a fortnight or 3 weeks after recovery. On this account, experimental protective inoculations undertaken in 1883 in Italy by means of subcutaneous injections of a virus as pure as possible, gave negative results (Nosotti).

**Symptoms in Sheep.**—In sheep and goats the manifestations of the disease are usually confined to the feet. The inflammation attacks the edge of the coronet, and extends to the cleft of the hoof, or, behind, to the plantar cushions. The coronet is swollen, reddened, and exudes a serous fluid. The affected animals become lame and lag behind the herd. We may sometimes find aphthæ on the toothless edge of the upper jaw, upper lip, gums of the inferior maxilla, and pudenda. Among sheep, sucking lambs become most gravely affected, and frequently die in a very short time under symptoms of severe gastric derangement. In full-grown animals the course of the disease is usually acute and benign. The affection runs its course much more slowly in a flock of sheep than in a herd of cattle or in a drove of pigs. The period of incubation is from 2 to 8 days.

The chief complication is the malignant form of foot disease, which appears to be a mixed infection of aphthous inflammation of the hoof and pararitium. Its presence gives rise to symptoms of erysipelas, lymphangitis, cellulitis, serous decomposition of the secreted horn, formation of abscesses and suppurative undermining of the hoof, separation of the horn at the coronet, shedding of the hoof, purulent inflammation of the glands of the hoof, purulent arthritis, necrosis of the bones, and pyæmia. The chief predisposing causes are: moisture, dirt, over-exertion from long journeys, excessive wear of the hoofs from bad roads, injuries from travelling over stubble-fields, etc.

The disease may be mistaken for ordinary traumatic eczema of the skin of the hoof, which, however, is never contagious,

shows no aphthæ, and runs a more chronic course. Hess states that he has observed a contagious, malignant inflammation of the hoof which is independent of the foot and mouth disease, and is caused by the *bacillus necrophorus*.

**Foot and Mouth Disease in Pigs.**—Here, also, the disease manifests itself more frequently on the feet than in the mouth. The period of incubation varies, on an average, from 36 hours up to 3 days, with a minimum of 24 hours. The disease appears to be uninfluenced by breed. The changes are similar to those in sheep. The chief symptoms are the vesicles and the inflammation of the coronet, with consequent lameness. We find in pigs the same complications as in the malignant foot disease of sheep, especially among pigs which are driven in herds. In the oral form of the disease, to which sucking-pigs are especially predisposed, the eruption of aphthæ spreads to the snout, tongue and palate, in the form of vesicles the size of a pigeon's egg up to that of a walnut. As causes of death from the malignant form, we may mention œdema of the lungs, broncho-pneumonia, hydrothorax, and purulent pleuritis. With regard to differential diagnosis, contusions of the plantar cushions from long journeys are of special importance.

**Infectious Aphthæ of Horses.**—In horses we find only the oral form of the disease. The infection is conveyed by cattle, goats, sheep and pigs. For instance, 1 case was caused by a horse licking a bullock which was suffering from foot and mouth disease (Bräuer). In other instances the disease was transmitted by a water-bucket. Many of the reported cases of foot and mouth disease in the horse should, however, be regarded with great suspicion; for evidently several of them were either those of stomatitis pustulosa contagiosa or stomatitis catarrhalis. Nevertheless, foot and mouth disease has been positively proved to have occurred in horses. The symptoms are the same as in cattle, and consist of fever, loss of appetite, eruption of vesicles on the oral mucous membrane, lips, and even on the nasal mucous membrane and skin, and sometimes catarrh of the stomach, intestines and bronchi. It is said that croupy processes occur more frequently in horses than in cattle.

**Infectious Aphthæ of Dogs** is a very rare disease; although stomatitis aphthosa and ulcerosa and inflammation of the skin between the toes and of the pads of the feet, have been seen in dogs which had come in contact with infected cattle or pigs, or which had been kept in infected stalls.

**Infectious Aphthæ of Cats and Rabbits.**—Vesicles and ulcers on the mouth and tongue, upper lip, and anterior part of the head, with fever, vomiting, and lameness, have been observed in cats and rabbits.

**Infectious Aphthæ of Birds**, which very seldom occurs, manifests itself by vesicles on the tongue, pharynx, nasal mucous membrane, conjunctiva, comb, and feet, including the web between the toes. Experiments by Kitt and Schindelka in the transmission of the disease to birds gave negative results.

**Transmission of Mouth Disease from Cattle to Man**

is by no means rare, and is more frequently seen by veterinary surgeons than by physicians. Children are specially liable to become infected by drinking unboiled milk from cows suffering from foot and mouth disease, and sometimes die from the consequences of the infection. Transmission may take place by butter and cheese, through wounds on the hands and arms, and indirectly by intermediate bearers. The symptoms of the disease in man consist of fever, disturbances of digestion, eruption of vesicles on the face (lips and ears), fingers, arms, chest, mucous membrane of the mouth, pharynx (angina) and conjunctiva, and diarrhoea accompanied by vomiting, which may cause the death of young persons. It appears that the disease cannot be transmitted by eating the flesh of infected animals. It is possible that the severe skin affections which have appeared in some years (1883-84, for instance) in children after vaccination, were really foot and mouth disease and not cow-pox.

We take the following brief notes on the transmission of foot and mouth disease from animals to men, from the very rich literature which exists on the subject. Sagar states that in 1764 several people became infected in Moravia. In 1778 all the inhabitants of a monastery had an attack of vesicles in the mouth after drinking milk. Berber (1811) reports similar cases in the department of Rhône in France; and Tilgner (1834) in Germany. Hertwig, Mann, and Villain in 1834 carried out the experiment in their own persons of drinking, for 4 consecutive days, milk warm from a cow which was suffering from a severe attack of foot and mouth disease. All 3, especially Hertwig, became ill on the second day with fever, which was followed by an eruption of vesicles on the mouth, fingers, and hands. Schneider observed, in 1840, in men violent abdominal pains, vomiting, and bloody and copious diarrhoea after drinking the milk of a cow whose udder was affected by apthæ. Butter and cheese have also been proved to be dangerous. From eating cheese, a male servant suffered from the oral form of foot and mouth disease and nervous fever; a peasant woman died of malignant mouth disease; and whole families became ill. During an enzootic of the foot disease in 1845, Gierer saw apthous affections of the mouth in children. Hulin states that out of 1,000 inhabitants of a village, 23 died of infective apthæ. Holm saw vesicles on the nipples of a woman who was giving milk to an infant, and Guilmont found in 2 girls apthæ on the mouth and hands. Zürn reports an instance of death from drinking the unboiled milk of an infected cow, in the case of a child whose gastro-intestinal mucous membrane showed numerous vesicles and ulcers *post mortem*. According to reports of Prussian veterinary surgeons in 1874, many people became ill from drinking butter-milk. Demme (1876) saw apthous stomatitis in twins, 1 of whom subsequently died, and the mother also became affected by the same disease. [Stang (1882-84) saw 2 women with apthæ in the mouth, nasal cavities, space between the fingers, and on the arm. In the district of Breslau (1885) severe cases occurred in children. One child 13 years of age had high fever, violent conjunctivitis, and apthæ in the mouth; and another 9 months old suffered from diarrhoea and vomiting, and died. Esser saw 4 children affected by vomiting, headache, pains in the limbs, and great weakness. Haarstick, von der

Ohe, Bührmann, Eggeling, Noack, Weigel, Walther, Hartenstein and others, report similar cases of stomatitis aphthosa, diarrhoea, etc.

Fröhner described, in 1880, a case in an adult of transmission of the disease by fresh butter. Symptoms of fever appeared after 12 hours, and in 24 hours the under lip and chin were swollen and covered with vesicles, which were also found on the tip of the tongue. In 3 days' time the ears became covered with vesicles, and erysipelatously reddened. Ptyalism and swelling of the parotid and sublingual glands were also present. The lesions healed in 12 days. In 1889, Weissenberg described a case in Berlin of a three-year-old boy who suffered from the disease, with symptoms of headache and pains in the limbs, considerable itching of the skin, vomiting, diarrhoea and rigors, with an outbreak on the third day of numerous vesicles on the lips, cheeks, tongue, tip of the nose and fingers. The entire eruption healed in 10 days. Siegel (1891) observed among several hundreds of people in the neighbourhood of Berlin a mouth epidemic, in the course of which 36 people died. He connected it with foot and mouth disease of cattle (?).

## VARIOLA.

Variola in general—Variola of man—Sheep-pox—Cow-pox—Horse-pox—Swine-pox—Variola of goats—Variola of dogs—Variola of birds.

**History.**—The variola of our domestic animals, like that of man, has been known for a very long time. Sheep-pox and small-pox of man are probably the only independent kinds of variola and probably have originally come from the East. We may point out that epidemics and epizootics spread from east to west. Animal variola appeared for the first time in 1275 in England. Rabelais in 1578 was the first to describe it in France, in which country inoculation had been introduced at a very early period. Ramazzini reports its occurrence in Italy during 1691; and Stegmann, in Germany during 1698. Bourgelat recognised its contagiousness in 1763. The disease was spread to an extraordinary extent by protective inoculation, which was universally introduced at the end of the eighteenth century (1770 in Germany); by the importation of foreign, especially, Spanish sheep; and by improvements in the breeding of sheep. Thus in 1819 more than 1,000,000 sheep died of variola in France; and in 1823 nearly half a million in Austria. By the help of the regulations for the suppression of epizootics, Germany is almost entirely free from sheep-pox which still rages in Russia, Hungary, France, England,\* and other countries.

\* There has been no sheep-pox in Great Britain since 1862.—TR.

Cow-pox has also been known in England for many centuries. Jenner in 1796 was the first to transmit it to man. He also proved that it conferred on men immunity to small-pox. Since that time, vaccination has become gradually established everywhere.

Horse-pox seems to have been of more frequent occurrence in former times than at present. Sacco and Jenner at the commencement of last century state that they have frequently seen it. Jenner considered horse-pox to be the starting point of cow-pox. Röhl states that horse-pox during 1855 in Vienna often occurred among young remounts. According to Bouley it is found more frequently than cow-pox in the neighbourhood of Paris. No case of horse-pox has been observed in Germany for several decades. In 1838, Hertwig inoculated himself and others with horse-pox, and thus produced on the hands and arms an eruption which closely resembled cow-pox. In former times, many cases occurred of blacksmiths becoming infected from shoeing horses which had horse-pox localised on the skin of the back of the pasterns. From this infection these men became immune to small-pox.

**Occurrence.**—Variola occurs in most of the domestic animals, such as sheep, cattle, horses, dogs, goats, pigs, as well as in man. It has also been observed in monkeys, camels, and buffaloes. It appears that variola is not an independent, primary disease in all species of animals. Old observers, such as Turner, Leroi, Ceely, Sunderland and Thiele, pointed out the genetic connection between cow-pox and small-pox. Roloff and Bollinger held the opinion that cow-pox derived its origin from small-pox. In fact, Bollinger maintained that small-pox and sheep-pox were the only two essential forms of variola; and that all the other kinds of variola (those of horses, cows, pigs, goats, and dogs) were adventitious forms of variola, which were respectively carried from man or from sheep to these other animals. The close relationship of these various kinds of variola is proved by their reciprocal power of transmitting the affection, and of conferring immunity. Their respective identity with small-pox is amply demonstrated by the frequent interchanges made between small-pox and animal variola.

Chauveau, having failed to set up variola in cows from small-pox, disputed the connection between cow-pox and small-pox; and expressed his opinion that, although both kinds of variola may have had a common origin, cow-pox



is not mitigated small-pox. On the other hand, Fischer, Pourquier, Ducamp, and others have succeeded in transmitting small-pox to cattle.

**Etiology.**—Variola is a contagious infective fever which manifests itself as an acute, typical exanthema. The contagium is both fixed and volatile. It is contained in the contents and crusts of the pustules, blood, secretions, excretions, expiratory air, and exhalations from the skin. In cattle it is usually absorbed by the organs of breathing or from the skin, when pustules are present. In the case of sheep-pox the virus can be carried for a distance of 200 metres. Chauveau has proved that the infective matter is contained in the solid and not in the fluid constituents of the lymph. Although the exact nature of the contagium has not been demonstrated, there is hardly any doubt that it is a bacterium. Zürn, Hallier, Cohn, Weigert, Klein, Toussaint, Semmer-Raupach, Plaut, Guttmann, Pfeiffer, Ruete, Guarnieri and other observers have attributed the disease to the action of micro-organisms. Guttmann and Grigorjew cultivated from the lymph a micrococcus, the *staphylococcus cereus albus variolæ* (*micrococcus vaccinæ*). Ruete believes that certain very motile diplococci are the cause. Pfeiffer repeating the investigations made by previous observers, has called attention to the existence in the plasma of peculiar spherical formations, which have a homogeneous capsule and a finely granulated nucleus. They are from 20 to 30  $\mu$  long, sometimes enclose several protoplasmæ, and are stated to break up into spores (sporoza). Nothing further is known respecting their connection with this disease. Van der Loeff, Guarnieri and others found in the plasma, motile corpuscles which appeared to be rhizopoda belonging to the family of the proteidæ and which were present only in the contents of the pocks. These observers state that these corpuscles are closely related to the virus of the variola.

The contagium of variola remains virulent for a considerable time; in fact it may continue so for some months when it is protected against decomposition. The contagium of sheep-pox remains active even in sheds for a month to 6 weeks, and is destroyed by putrefaction, high temperature, carbohc acid, etc. An attack confers immunity from subsequent ones. This immunity may be regarded as permanent, considering the short life of sheep.

**General Symptoms.**—The course of an attack of variola is usually divided into 5 stages, namely; incubation, initial period, eruption, suppuration, and exsiccation.

1. The *period of incubation* is about a week on an average.
2. The *initial stage* (*stadium prodromorum*), which lasts a day or two, is marked by fever, catarrhal affection of the mucous membranes, and erythema of the skin.
3. The *stage of eruption*, which continues from 6 to 8 days, manifests itself by the sudden appearance of red spots like flea-bites or pimples, which turn into firm red nodules the size of the head of a pin, and are surrounded with a red ring. They are chiefly found in the neighbourhood of the orifices of skin follicles. After a few days the tops of the nodules become pale and turn into bluish-white vesicles, which, especially in cases of inoculated variola, are depressed in the centre and are filled with limpid serous fluid. At this period the pocks are at their highest state of maturity, and are then most suitable for inoculation purposes.
4. During the stage of *suppuration*, which lasts 2 or 3 days, the contents of the vesicles become turbid and purulent; the vesicles change into pustules; and the depression in their centre disappears. The temperature, which had decreased during the stage of eruption, again rises.
5. During *exsiccation* the pustules gradually dry up into at first yellowish, and later on, dark-brown crusts or scales, which become detached and leave behind white shining cicatrices or brownish-red spots. The process of exsiccation goes on for 3 to 5 days.

Differing from the foregoing normal course of variola, several of the pocks may run together, and form large pustules (*variola confluens*), or the eruption may be complicated with a hæmorrhagic diathesis (*variola hæmorrhagica*), with gangrene of the skin (*variola diphtheritica*) in many cases.

### **General Remarks on the Anatomy of Variola.**—

If we follow the development of variola under the microscope, we shall see at first that the cells of the mucous layer above the papillary body swell and form here and there pale flakes which contain no nuclei. Afterwards, the papillary body transudes a serous fluid, and all the epithelial cells of the stratum mucosum dissolve with the exception of a few, which become drawn out into trabeculæ, and threads by the accumulated exudate. The vesicles thus form, between the papillary body and the horny

layer, a cavity which is filled with fluid, and has trabeculæ and threads intersecting it. At the same time the papillary body and the cutis lying under it become inflamed, swollen, and infiltrated with round cells. The pustule develops from the vesicle by an abundant immigration of leucocytes from the papillary body into the fluid of the vesicles, and by the purulent breaking down of the septa. Healing takes place by the exsiccation of the pustule, and by the absorption of the cellular infiltration under the crust. The epidermis becomes replaced from the remaining tissue and from the edges of the sore.

The origin of the depression in the centre of the vesicle is explained in different ways. Some say that it is caused by the hair follicles and the orifices of the sweat glands, which lie in the centre of the pock\*; others, that the trabeculæ in the interior of the vesicle prevents the centre of the pock from rising up. It is also ascribed to the inflammatory infiltration of the skin at the periphery of the pock.

**Variola of Men** has been much longer known than that of animals. It was recognised in ancient times, and spread, especially in the Middle Ages, over Western Europe, where, even during the eighteenth century, there was an annual mortality from it of nearly half a million. The practice of the inoculation of small-pox is exceedingly old, and has long been current in India and China. From thence it came through Asia to Constantinople, where Lady Wortley Montagu had her son inoculated in 1717. From Constantinople the inoculation of small-pox was first introduced into England, where Jenner proposed vaccination in 1798. From England it was brought to the Continent.

#### SHEEP-POX.

**Etiology.**—Owing to its well-marked epizootic character, sheep-pox is of greater economic importance than the variola of any other domestic animal. The infective matter is fixed as well as volatile, and possesses such vitality that it will continue virulent in sheds for as long as 5 or 6 months. Animals which have had the disease, whether naturally or by inoculation, will remain capable of transmitting it for 6 weeks after recovery. It can, however, be speedily and effectually destroyed by diluted hydrochloric acid (Nocard),  $1\frac{1}{4}$  per cent. solution of carbolic acid, 5 per cent. solution of chloride of zinc or quinine, 10 per cent.

\* The idea here is that the hair follicle or sweat gland being firmly connected to the underlying true skin, prevents the epidermis at its external attachment from rising under the influence of the pressure exerted by the fluid contained in the vesicle; and that, in confluent variola, the said connection with the true skin becomes destroyed.—TR.

solution of permanganate of potash (Grünwald), and by all the stronger antiseptics. The contagium chiefly spreads by infected, recently recovered, or inoculated sheep; or by intermediate bearers, such as shepherds, dogs, clothes, wool, skins, manure, fodder, railway-trucks, etc. During the course of the disease the milk is very virulent. Almost all ordinary sheep are susceptible to the contagium with the exception of newly-born lambs, which remain immune for a short time, or may even continue permanently immune if the ewe had been inoculated shortly before lambing. On the other hand, it is possible that lambs may be suffering from variola at the time they are born, or the fœtus may die in the uterus in consequence of the infection. Algerian sheep (Chauveau) and Brittany sheep (Nocard) are more or less immune.

The variola of sheep may be transmitted either naturally, or by inoculation to cattle, goats, pigs, horses, and men. Schmidt and others have described a case of ordinary infection in man. Also, variola of man and cow-pox can be transmitted to sheep. The direct introduction of human variola into the blood in sheep produces a general eruption of variola (Küchenmeister and Tappe).

**Statistics.**—During 1886, in Germany, 4,792 sheep became infected with variola, the chief centre of the disease being East Prussia. Germany has since then remained free from this disease, except in 1889, when a sporadic attack, which was quickly extinguished, occurred in 45 animals. In Austria, 18,000 sheep became infected during 1888. From 1877 to 1887, 0.5 per cent. of all the sheep in Austria were affected, with a mortality of one-third. In 1887, 17,000 sheep suffered from it in France; in 1889, 10,000 in Hungary; and in the 4 years from 1887 to 1890, nearly 150,000 sheep in Roumania.

**Symptoms.**—After a period of incubation of, on an average, 4 to 7 days, with a minimum of 2 or 3 days, the animal becomes feverish, has rigors, is depressed and weak, hangs its head, and ceases to feed and ruminate. The temperature rises to  $41^{\circ}$ – $42^{\circ}$  C.; the pulse is frequent, and the respiration accelerated. The conjunctiva is greatly congested and there is a slight serous discharge from the eyes and nose. A day or two later, red spots and subsequently red pimples appear in rapid succession on those parts of the skin which are free from wool, or which are only slightly covered with it, especially the head, neighbourhood of the eyes, nostrils, mouth, inner surface of the fore-limbs and hind-quarters, chest, abdomen, and lower surface of the tail. Less frequently the exanthema appears on

the woolly parts of the body, and in some rare cases on the mucous membrane of the mouth and pharynx. As a rule, the eruption is not simultaneous; consequently different stages of the process may be seen close together on the same animal.

On the fifth day after the eruption, the pimples become pale on their summits and surrounded with a red areola. When the pocks lie in great numbers close to one another, as on the head and about the eyes, the neighbouring skin becomes greatly swollen. At this time the temperature falls, and a few days afterwards the vesicles increase in size; being sometimes raised, at other times flat. They then contain a limpid, colourless, or orange-coloured fluid. On the sixth or seventh day after the eruption, the pocks become ripe.

From this period the contents of the pocks become more and more turbid, and the vesicles turn into pustules; this change being accompanied by severe general catarrhal fever. The temperature again rises; the mucous membrane of the eyes, nasal cavity, pharynx, larynx and bronchi becomes catarrhally inflamed; a muco-purulent secretion flows from the eyes and nostrils; the animal slavers, regurgitates, coughs, and suffers from difficulty in swallowing and breathing; diarrhoea sometimes appears; the head becomes greatly swollen; and the exhalations from the skin have a very bad odour. The pustules wither and dry up, the epidermis contracts, and the crust, which was at first yellow, assumes a dark-brown colour, and finally falls off, leaving a small depression in the skin, or a cicatrix, on which little or no wool will grow.

Under these normal circumstances the course of the disease lasts for about 4 weeks.

**Course and Prognosis.**—The course of variola may present many departures from the *normal type* just described. In some cases we find a very mild or even *abortive form*, in which the pocks are few and discrete, and the fever is slight. In other cases the morbid process does not go beyond the development of pimples, which peel off after a few days. Lastly, the eruption is confined to vesicles, with only a slight effusion of lymph and without the formation of pustules.

In *variola confluenta*, which is a severe variety of the disease, several of the pocks run together so as to produce large pustules that give rise to extensive suppuration, great inflammation, swelling, and even gangrene of the skin, from which a foetid odour is often exhaled. There is at the same time high fever;

pocks are seen on the mucous membrane of the mouth, pharynx, larynx, bronchi, and even on that of the cornea ; the respective lymph glands are considerably swollen and sometimes suppurate ; the inflammation of the mucous membrane may sometimes assume a croupy character ; and croupy pneumonia becomes occasionally developed. In unfavourable cases, symptoms of septicæmia or pyæmia with metastases in the articulations, serous membranes of the brain, etc., may appear. Or the patients may die from pneumonic sequelæ or from asphyxia, caused by laryngeal croup. Sheep take a long time to recover from an attack of this kind of variola, which gives rise to great exhaustion and cachexia. They sometimes lose their entire fleece, and often become chronically lame or blind. The pustules leave behind deep-seated scars.

In *variola hæmorrhagica*, simultaneously with the appearance of the eruption, hæmorrhages break out everywhere on the skin, mucous membranes, and especially in the pocks themselves. This hæmorrhagic infiltration of the pocks leads to their gangrenous disintegration (*variola diphtheritica*). Among other symptoms, hæmaturia is also observed.

The duration of variola in a herd may be very short, or may be prolonged for months. The course of the complaint varies greatly in intensity. Only 2 or 3 per cent. escape infection, the mortality from which amounts to from 10 to 20 per cent. under ordinary circumstances, and to 50 per cent. or more under unfavourable ones. The prognosis is particularly unfavourable in cases of variola confluens and variola hæmorrhagica, and when the animals are either very old and weak or very young (sucking lambs). The percentage of losses may then be as high as from 80 to 90 per cent. Very hot or damp cold weather, bad food and unsanitary housing exercise an unfavourable influence on the course of the disease. Freshly-imported sheep become more severely infected than home-bred sheep, acclimatised sheep, and those which have been previously attacked and are consequently immune.

Not counting the direct losses from death, we find that there is a fairly large indirect loss from reduced production of wool, decrease in bodily weight, frequent abortion, and chronic morbid sequelæ.

**Differential Diagnosis.**—Sheep-pox may be confused with pustular eczema and urticaria of the head, especially when they are produced by certain fungi, such as rust-fungi and mould-

fungi. If sheep-pox has not been observed for some years in a district, we may conclude that such sporadic pustular exanthemata are not variola.

**Therapeutics.**—The treatment of sheep-pox, like that of all other acute exanthemata, is purely expectant and dietetic. In complicated cases it may be necessary to apply a symptomatic treatment which can be carried out only with difficulty, or not at all, when a whole herd has become infected. Inoculation and veterinary police measures are the most important factors in *prophylaxis*.

**Inoculation of Sheep-pox (Ovination).**—*Inoculation* against sheep-pox, which, during the first half of last century and up to the end of the sixties, was practised as a prophylactic measure, even when no outbreak was threatening, has been found to be in every way objectionable. It not only artificially maintains permanent centres of the disease, but also spreads it, as has been well proved, from these foci to neighbouring healthy districts. In several countries, such as Prussia and Austria, the dissemination of sheep-pox went hand in hand with inoculation.

Inoculation is, however, to be recommended when the disease has already broken out in a herd; for it not only shortens the stay of the epizootic, but also produces, as a rule, a milder and more local affection than ordinary sheep-pox. The mortality is often *nil*, and is usually not more than 2 per cent. Only exceptionally, and under very unfavourable conditions, does it amount to 10 per cent. Inoculation may be practised in healthy sheep which are in the neighbourhood of a severely infected flock, and are in great danger of catching the disease.

**Technique of Inoculation.**—The inoculation material, or “ovine,” must be taken only from those sheep in which the course of the disease is benign, and the pocks fully ripe. The lymph should be as bright and clear as water, without any trace of turbidity or purulence. Consequently it is usually taken from the inoculated sheep 10 or 12 days after the inoculation, or 6 or 8 days after the eruption has appeared. The inoculated sheep should be segregated, and the sheep which are to be inoculated should not come into direct contact with the sheep from which the inoculation material is to be obtained, lest natural infection may take place simultaneously with artificial (inoculated) infection. The site of the inoculation is either on the inner surface of the ear, about 4 cm. from the tip (which position is not without danger owing to the proximity of the eyes), or, better still, on the lower surface of the tail, at about 10 to 12 cm. from the anus. In the latter case the

animal must be put down in order to perform the operation, which may be done with either a narrow and pointed inoculation needle, having a spoon-like hollow, or with an inoculation lancet. If time be not pressing when inoculating a whole herd, it is well to perform an experimental inoculation with from 6 to 12 sheep. The symptoms in inoculated sheep are those of a mild and regular attack of variola, with local manifestations restricted to the site of the inoculation and very slight general disturbance. The pocks become ripe on the tenth day after the inoculation. In exceptional cases the pocks do not appear on the site of the inoculation itself, but on the parts adjoining it. The eruption caused by the inoculation is followed, only in very rare cases, by a general eruption (secondary pocks). The after-treatment of inoculated sheep consists in protection against unfavourable conditions of weather and attention to diet. After 10 or 12 days the flock should be examined, and those animals which have not "taken" should be inoculated a second time.

In recent times several experiments have been made with attenuated (mitigated) inoculation material. Peuch attempted this by diluting the lymph with water (1 to 50-150); Nocard and Mollereau, by diluting it with water containing oxygen; Semmer-Raupach, by heating it to 55° C.; and Toussaint and Plaut, by cultivating it in nutrient solutions. Pourquier has found that inoculation with a pustule which has become developed from a previous inoculation with the ordinary virus of sheep-pox on the eleventh or twelfth day, confers immunity without giving rise to the eruption. He recommends the inoculation to be made at the end of the tail.

### Cow-Pox.

**Etiology.**—Jenner's original idea that cow-pox sprang from horse-pox is nowadays held by very few. Even in former times, Turner, Leroy, Ceeley, Sunderland, Thiele and others, pointed to the connection between small-pox and cow-pox. According to Bollinger, however, the source of cow-pox is not small-pox, as was formerly supposed, but human cow-pox (*variola vaccina*). This view is strengthened by the fact that enzootics of cow-pox often occur at the time (spring) when children are vaccinated; that cows have frequently been infected by human beings, especially by milkmaids who were recently vaccinated; and that human vaccinia can be easily transmitted to cattle.

The contagium of cow-pox differs from that of sheep-pox, by being fixed and not also volatile; consequently, direct contact of the contagium, as in milking, with the injured skin is always necessary for the infection. According to Chauveau, Warlomont, Hugues, Straus and other French investigators, intravenous, intraocular, and subcutaneous inoculations confer immunity without producing either a local or a general attack of variola.



The transfusion of vaccine blood from calf to calf gives immunity without any visible affection (Reynaud). Cow-pox can be transmitted to sheep, goats, horses and men; and human variola and vaccinia to cattle. According to Sunderland, Dinter, and Woodville, the direct inoculation of human variola or vaccinia generally confers immunity, and is accompanied only on rare occasions by an eruption.

**Statistics.**—From 1825–1868, 241 cases of cow-pox, and from 1873–78, 100 cases were reported in Württemberg. In 1874, 374 cases; in 1877–78, 1,037 cases; and in 1888–89, 878 cases occurred in Denmark. As a rule, cow-pox is a rather rare disease.

**Symptoms.**—Cow-pox is chiefly found in young cows which have been recently put to milk. The eruption is generally confined to the teats and to the adjacent parts of the udder. Fever is either absent or only slightly marked. The development of the pocks takes place in characteristic and well-marked stages. There is sometimes a slight disturbance of the general health of the animal, which is best shown by decrease in the quantity of the milk. The milk is thinner and of lighter specific gravity than usual. The implicated teats are somewhat sensitive, slightly swollen, and show, particularly at their base, isolated pink pimples, which subsequently change into vesicles. These pimples number as a maximum from 20 to 30, and vary in size from a lentil to a pea. The colour of these small and large vesicles varies according to the condition of the skin. They are bluish-white and nacreous when the skin is white and very fine; red and have a metallic lustre when the skin is light-coloured and thin; leaden-coloured and possessed of a metallic sheen when the skin is dark and thin; and cream-coloured and lustreless when the skin is thick and free from pigment. The vesicles have a depression in their centre; they are elliptical in form on the teats, and circular on the udder; and are surrounded by a red ring if the skin is not pigmented, and by a tumid, swollen and firm margin. They become matured about the tenth day, and are then the size of a bean. From that time they quickly become purulent, pointed, and dry up into a dark-brown, shining crust, which falls off in about 4 days and leaves a cicatrix. The pocks are frequently torn off during the process of milking. The duration of the eruption is about 21 days. Prognosis is so favourable that treatment is superfluous. Only in rare cases the eruption spreads over the whole body, and may then be found also on the head, inner

surface of the thighs, buttocks, back, throat, chest, etc. In bulls it has sometimes been seen on the scrotum.

The course of the disease in the same animal is characterised by successive attacks, so that the respective pocks show different degrees of development. The affection spreads slowly from animal to animal in a shed, until all the cows, with very few exceptions, become attacked. Bulls, oxen, and young cattle become less frequently affected than cows. They sometimes receive the infection from the litter. Cow-pox usually lasts for several weeks in a shed.

**Differential Diagnosis.**—The eruption of cow-pox may be mistaken for that of foot-and-mouth disease, rinderpest, eczema of the pasterns from feeding on distillers' wash, mercurial poisoning, which may be set up by the use of mercurial ointments for inflammation of the udder, and false cow-pox or varicella (chicken-pox), the vesicles of which vary in size from a pea up to a cherry, and, on drying up, leave behind them thin, paper-like crusts. In this respect, confusion may also exist with so-called variola verrucosa, which consists of a multiple non-contagious eruption of papilloma on the teats and which gradually disappears after having been present for months.

**General Notes on Animal Vaccination in Man.**—Since the time of Jenner (1798) the "original" lymph, namely, the true lymph of cow-pox or "vaccine" has been used for the vaccination of man. As, however, cow-pox is comparatively rare, and as its vaccine can therefore be obtained only with difficulty, "humanised" vaccine, that is, vaccination from man to man, was employed later on. On account of the possibility of the simultaneous transmission of human diseases, such as syphilis, animal vaccination has been recently re-introduced. Vaccination institutes for the purpose of animal vaccination were at first established in Milan, then in Paris (1864), Brussels (1868), and later on in all countries and large towns. For its cultivation, calves 5 to 12 weeks old are generally used. The technique of the vaccination is very simple. The calves are fixed in the left lateral position on a table with the right hind leg drawn perpendicularly upwards. The skin from the symphysis pubis up to the umbilicus, and laterally to the fold of skin at the stifle, is shaved and carefully disinfected. Then, either "incision" inoculation or "surface" inoculation is performed: namely, we make either cross incisions about 2 cm. long, or we inoculate the entire surface by means of from 50 to 200 punctures, and place on the incisions or on the punctures mature lymph from a previously inoculated calf. The vesicles become mature in 4 or 5 days, and the lymph contained in them is used for the vaccination of human beings and for further inoculation of calves. The inoculation material is obtained by means of a clamp forceps, with which the

lymph can be easily squeezed out of the vesicle. One calf yields from 1,000 to 3,000 doses. The vaccine may be preserved in various ways. Capillary tubes are not to be recommended, because the vaccine, when kept in them, frequently becomes inert in as short a time as a week. A better plan is to keep the vaccine in a dry condition, which we may do by scraping off the lymph and crusts, drying them, and placing them between two glass plates, which are closed up with paraffin. In this way the lymph keeps well for months, and even years; or the lymph may be rubbed down in glycerine with the addition of an antiseptic, and is then kept in capillary tubes. As an antiseptic, Röpke recommends: thymol, 0.1; spiritus, 0.5; glycerine, 100 grammes, or acid salicyl, 0.25; aqu. destill. glycerine aa, 50 grammes.

Animal vaccine gives quite as good results, as regards the effect and development of the exanthema, as human vaccine, to which it is greatly to be preferred. Animal vaccine can be produced in any desired quantity, and its use is free from the danger of transmitting human diseases. The chance of transmitting tuberculosis by its employment need hardly be considered; for tuberculosis is very rarely met with in young calves, and no proof has yet been given that it can be transmitted by vaccination. Besides, we can obviate any such risk by a previous veterinary examination, and by the employment of tuberculin. We need hardly say that only healthy and perfectly unsuspected animals should be selected for the inoculation.

### HORSE-POX.

**Etiology.**—Formerly horse-pox was much more common than at present, and is now exceedingly rare. It occurs apparently only in certain districts, for instance that of Paris, according to Bouley. It is probable that small-pox is the true origin of horse-pox, which is consequently identical with cow-pox and which can be transmitted to cattle and mankind. Inoculations of horse-pox in cows produces pocks similar to those of cow-pox and capable of producing the same protective effect in men. According to Hertwig and Pingaud, artificial as well as natural transmission of horse-pox to men (shoeing-smiths and grooms) produces in them an exanthema similar to that set up by cow-pox. Bollinger considers that the predilection shown by the exanthema of horse-pox to attack the skin behind the pastern is due to the fact that that part is peculiarly liable to the fissures and other lesions of cracked heels and grease. In our opinion this liability may be more justly ascribed to the fact that that site is frequently touched by recently vaccinated shoeing-smiths and grooms, who probably often act as intermediate bearers of the infection.

With respect to this mode of transmission, the skin at the back of the pastern of the horse is analogous to the udder of the cow.

Horse-pox now rarely becomes general, a fact which Chauveau

observed when he made his inoculation experiments with vaccine, intravenously and subcutaneously, by inhalation and by feeding. Warlomont and Pfeiffer obtained similar results.

The variolæ of sheep, cows, and man can be respectively transmitted to horses. Berger observed on two occasions natural infection of horses by sheep-pox.

**Symptoms.**—The first symptoms are increased temperature, debility, and loss of appetite. We then notice on the flexor surface of the pasterns, first of all, an erythematous exanthema which consists of congestion and swelling of the skin, and may spread upwards to a greater or less extent. At the same time the animal becomes lame, and, if made to move, stumbles all over the place, and lifts his feet high off the ground. On the inflamed part we find vesicles and pustules which soon burst and leave the skin highly congested, painful to the touch, considerably swollen, and covered with a viscid secretion. The surface of the affected part gradually dries up, or becomes covered with a scab, and the lesions heal in a few weeks. In rare cases the exanthema attacks other parts, especially those portions of the skin of the head in the neighbourhood of the lips and nostrils which are only thinly covered with hair ; and the mucous membrane of the nose, mouth, and conjunctiva.

**Differential Diagnosis.**—Although the occurrence of horse-pox as a distinct disease is well established, its eruption has been frequently confused with that of other diseases, especially with the exanthema of stomatitis pustulosa contagiosa, which closely resembles it, except as regards the site of the attack. In France the two diseases are regarded as being identical. The fact that the eruption of stomatitis pustulosa can be readily transmitted by inoculation, even to cattle, makes a mistake all the easier. In our opinion, the two diseases have, etiologically, nothing in common. Grease and cracked heels may be confused with horse-pox, but can be easily differentiated from it by the fact that they do not manifest the typical course of variola, and that they cannot be transmitted by inoculation. The vesicular eruption on the genitals may possibly be mistaken for horse-pox.

#### SWINE-POX.

**Etiology.**—Swine-pox seems to be obtained chiefly from human beings (by means of bed straw), or from sheep, as for

instance, on account of the animal occupying the same stall as an infected sheep. Young pigs are more often affected than older ones. Swine-pox can be transmitted experimentally to goats (Gerlach), and back from them to pigs. It can also pass in the ordinary way to men. In Hungary 517 pigs became affected with variola during 1891.

**Symptoms.**—Usually a general eruption breaks out over the entire body. There is fever, debility, loss of appetite, and congestion of the mucous membranes. Red spots (like flea-bites) appear on the head, throat, back, chest, inner surface of the fore arms and thighs, abdomen, etc. These spots change successively into pimples, vesicles and pustules, which finally dry up, and leave black, concave, circular crusts. In exceptional cases we may notice an eruption on the oral mucous membrane, conjunctiva, cornea, and mucous membrane of the stomach and intestines. Often the disease varies greatly in intensity. In a few cases the eruption is accompanied by lobular, catarrhal broncho-pneumonia, ending in death (Csokor). Swine-pox has been frequently confused with urticaria, pustular eczema, and other diseases.

#### VARIOLA OF GOATS.

The rarity of caprine variola accounts for the fact that we find mention of only a few cases in veterinary literature. In Norway, 264 cases occurred during 1890. Brémond states that the variola of goats is a disease peculiar to these animals, because it cannot be transmitted to sheep by inoculation. It is also stated that goats are immune to the variola of sheep. The course of the eruption in goats may be the same as in sheep, or may be similar to the pustular udder affection of cows. Hertwig states that goat-pox is characterised by its course being marked by successive attacks.

#### VARIOLA OF DOGS.

Dog-pox can be transmitted experimentally from man to dog (Dupuis and others). In rare cases this may also occur accidentally (Weiskopf). We must view with suspicion many of the cases mentioned in veterinary literature; because this disease may have been confused with the pustular exanthema of distemper or perhaps with mange and epizootic aphthæ. In any case, dogs are very slightly predisposed to variola.

The supposed cases of variola in **birds** were probably those of gregarious epithelioma. Even experimentally, variola cannot be transmitted to birds (Günther, Hütrel, d'Arboval, Rayer, Plaut, and others).

RINDERPEST (*Cattle Plague*).

**History.**—Although rinderpest was not known in classical times, it has probably existed for ages in the steppes of Eastern Europe and Central Asia. Its history is as ancient as that of the inhabitants of those regions. Rinderpest was brought for the first time to Western Europe by the migration of nations in the fourth century, and by the migration of the Huns from Central Asia, of the Alans from the Volga, of the East Goths from Southern Russia, and of the West Goths from Northern Hungary. Since then it has remained as a permanent resident in Western Europe. After its first appearance it was continually disseminated afresh over the countries of Europe by the many wars which took place, especially in the ninth century during the reign of Charlemagne, when Germany, in particular, suffered great losses in cattle. At the commencement of the thirteenth century, rinderpest devastated Eastern and Central Europe during the invasion of the Moguls. Rinderpest caused great havoc through Europe during the Northern War, the War of Succession, and the Seven Years' War in the eighteenth century. The first great epizootic took place in the years 1709 to 1717, and spread from Tartary over the regions of the Don and Volga to Moscow, Poland, Hungary, Prussia, Austria, Southern Germany, Switzerland, Italy, France, Holland, and England. It is stated that one and a half million of cattle perished during the years 1711 to 1714. Ramazzini was the first to give an exact description of the disease, which he compared to variola. During his time veterinary police regulations were first instituted against rinderpest. Another epizootic, which was described by Buniva, occurred during the years 1726 to 1734.

From the middle of the eighteenth century to the commencement of the nineteenth century, rinderpest was a permanent resident in all the countries of Europe, with the exception of Spain and Sweden, into neither of which any cattle were imported. During the years 1740–1750, the loss of cattle was estimated at 3,000,000 head. From 1745–1752, it amounted, in Denmark alone, to 2,000,000. The total loss in Germany up to the end of the eighteenth century was reckoned at 30,000,000 head; and in Europe, at 200,000,000 head. The ravages of rinderpest were the chief reason for the founding of veterinary colleges, which began to spring up at that period. Many of the descriptions made at that time by Bourgelat, Boerhave, Wolstein,

Layard and others, are still extant. Lutz states that up to the year 1783 about 1,000 publications on rinderpest had been brought out. At that period protective inoculation against rinderpest was practised for the first time.

Inoculation was introduced into England by Dodson in 1744 ; into France by Courtivron in 1745 ; and after that into all other countries.

Rinderpest destroyed between three and four millions of cattle during the year 1792 in Italy, into which country it had been introduced by oxen which were intended to be used as food for the Austrian army. From 1795 to 1801 it raged in Southern Germany ; and was widely spread by the wars of Napoleon (1805-1809), and by the wars of liberation (1813-1816). It invaded Europe from Moldavia and Wallachia during 1827-1828 ; and from Russia, Poland, and the Baltic provinces during 1830-1831 (Polish revolution). In 1844 rinderpest was introduced into Egypt from Roumania and Anatolia with a loss of half a million cattle. Russia lost one million cattle in 1844-1845. Prussia, and particularly Austria, both of which countries are on the frontiers of Russia, were visited by rinderpest nearly every year. For instance, Röhl states that Austria lost nearly half a million cattle during the years 1847-1864. The wars of 1866 and 1870-1871 caused a wide dissemination of the disease. During the latter war 70,000 died in France, 30,000 in Alsace-Lorraine, and 10,000 in Germany. The last serious invasion of Germany occurred in 1878-1879, during which period 2,500 cattle died in Prussia, making a gross loss of £100,000 ; and 350,000 in Russia. We may say, speaking generally, that rinderpest has disappeared at least from Germany, thanks to the prohibition of import and the strict execution of the epizootic regulations.

[Our exact knowledge of the occurrence of rinderpest in England dates from 1865, in which year this bovine scourge was imported from Revel by Russian cattle. After a loss of over £5,000,000 sterling, it was stamped out during the following year. It was again introduced from Russia into England in 1872, but being recognised, its spread was quickly stopped. Its last appearance in England was in 1877, on which occasion it caused a good deal of loss before it was annihilated. So-called murrain (Old French, *morir* = *mourir*) broke out among cattle in England during the years 1348, 1349, 1480, 1715, 1745, 1757, and produced great havoc. Williams was strongly of opinion that the bovine epizootic of the eighteenth century was rinderpest.

The latest invasion of rinderpest is that of South Africa, where it has caused immense losses.—Tr.]

**Bacteriology.**—Sanderson, Bristowe, Murchison, Beale, Semmer, Naczynski, Hallier, Klebs, Roschnow, Woronzow, Medwedski, Saweljeff, Metschnikoff, Gamaleïa, Sacharow, Tokishige, and many other investigators have occupied themselves in the bacteriological study of the infective material of rinderpest, which, according to Semmer, cannot be demonstrated by the present methods of staining and cultivation. Semmer is of opinion that the only bodies which can be regarded as the cause of the disease are very small corpuscles, in the condition of spores, which can be distinguished only with difficulty, and which cannot be cultivated. He says that they exist in numbers from 1 to 6 in the enlarged cell nuclei, and that they are frequently surrounded by a bright areola. Saweljeff cultivated as infective matter sporulating bacilli, which break up into micrococci and streptococci. The colonies on agar-agar were at first greyish-white, then lemon-yellow, and finally red. The bacilli could be stained with methylene violet. Inoculations with these cultures produced rinderpest. The bacilli themselves are very motile, and have rounded ends. When found in the blood they may be straight or curved, and can be seen only with difficulty when unstained. Saweljeff believes that former investigators have seen the same bacteria when they were in a different form of development. Metschnikoff describes a short bacillus of rinderpest with rounded ends which sometimes assumes the form of cocci, and which grows out into leptothrix-like threads. These bacilli are found chiefly in the ulcers of the abomasum, but are also met with in the blood. The cultures do not liquefy gelatine. Gamaleïa produced with pure cultures rinderpest in calves and guinea-pigs, but not in rabbits. Sacharow describes as specifically pathogenic a stout bacillus  $\frac{1}{2}$  to  $1\frac{1}{2}$   $\mu$  long; and Tokishige, a very small and short bacillus, the pure cultivations of which are stated to have produced the symptoms of rinderpest in experimental cattle.

**Pathogenesis.**—Semmer assumes, probably with justice, that emigration of the infective matter, which has not yet been positively identified, into the body takes place through the respiratory apparatus, from whence it gains entrance into the blood, and sets up a general infection in it, only after secondary changes have become developed in the digestive apparatus.

The infective matter is both fixed and volatile. It is contained in the secretions and excretions, namely in fæces, urine, saliva, tears, and milk; mucous membranes of the nose, mouth, and eyes; sweat; expired air; blood; and various organs of the body. The infection may take place either directly through affected animals, or indirectly by intermediate bearers, such as manure, litter, straw, hay, soil, skins, wool, flesh, clothes, railway-trucks, ships, butchers, cattle-dealers, smugglers, dogs, sheep, birds, etc. It is characteristic of the contagium of rinderpest that it can be carried only for a short distance by the air, and that this distance varies inversely with the dryness of the atmosphere, and is consequently shortest in summer



The average distance is] probably 25 paces. It has often happened that in summer the transmission of the infection has been prevented by a ditch which separated the infected animals from the healthy. It thus appears that the contagium is rapidly destroyed in air, and especially in dry air. On the other hand, it remains virulent for a comparatively long time in fluids, and in the solid tissues of the body. If the nasal mucus which is used for inoculation be hermetically sealed up, it will remain virulent for 6 weeks, and in a few cases even for 9 months. The infective matter remains active in sheds for 4 months and in hay for as long as 5 months. Flesh retains its virulence after it has been buried for 3 months, and manure which continued in a frozen condition throughout the winter, proved effective after it was dug up in the spring. The intensity of the infective matter is severest at the commencement of an epizootic. The virus is destroyed by desiccation, heating to a temperature of over 60° C., cooling it down to a temperature of - 15° C., putrefaction, and various disinfectants such as chlorine, sulphurous acid, carbolic acid, etc. It is easily destroyed by disinfectants.

**Occurrence.**—Rinderpest is an infective disease peculiar to ruminants. Besides cattle, it can be transmitted to sheep, goats, deer, buffaloes, yaks, antelopes, camels, gazelles, aurochs, etc., and also to pigs according to Penning and Pluning. As a rule, one attack confers life-long immunity. Opinions vary as to the original home of rinderpest, which according to Sergejew and Semmer is the black earth district of European Russia (Southern Russia, neighbourhood of the Black Sea, and the country of the Don Cossacks). This district has a layer of humus a foot deep and luxuriant vegetation. Other investigators state that it originated in Asia, Siberia, the Kirghiz steppes, Tartary, China, Persia, India, etc., and consequently outside of European Russia. We at least know for certain that rinderpest never becomes independently developed outside the Russian or Asiatic steppes, and that every epizootic of it in Europe has proceeded from those districts. Thus it was proved that the outbreak of rinderpest which took place in England in 1865, was caused by Russian oxen which had been imported by sea; and the epizootic in Switzerland in 1866, by Austrian oxen, the infection of which was traced to the introduction of the disease from Russia. For this reason, Germany and Austria, which are on the frontiers of Russia, are continually threatened with rinderpest. Formerly

the disease was spread by bullocks intended for slaughter during times of war ; but is now disseminated by the transportation of cattle, traffic in cattle, fairs, etc. Only in rare cases is it carried by skins, flesh, wool, butter, fat, etc.

**Symptoms.**—The symptoms of rinderpest are those of a severe, acute, infective disease, in which the digestive tract is the chief point of attack. The period of incubation is usually stated to be from 6 to 9 days. Roloff and others have, however, observed a rise of temperature in from 36 to 48 hours after infection. Raupach and Ravitsch found in their inoculation experiments, pronounced pathological changes in, respectively, 11 hours and 33 hours after inoculation. Semmer found the micro-organism in the blood and nasal mucus as early as 7 hours after inoculation. Taking into consideration that the first symptoms of the disease, of which increased temperature is one of the chief, may easily be overlooked ; we may estimate the period of incubation at from 3 to 7 days.

At first the temperature rises to  $41^{\circ}$ – $42^{\circ}$  C. and remains near that height with but slight remissions, until other symptoms become developed, when it falls, as is the case in all other infective diseases. The pulse is small and beats from 60 to 120 in the minute. There is great debility, decrease in the yield of milk, and loss of appetite. The coat loses its gloss and stands on end, and the muzzle is dry. The function of rumination becomes disturbed, and the animal may have slight shivering fits.

After these preliminary symptoms, well marked rigors come on ; the superficial heat is unequally distributed ; the breathing becomes accelerated ; and the visible mucous membranes assume a scarlet colour, or manifest scarlet spots on their surface. There is entire loss of appetite ; arrest of the function of rumination ; thirst ; constipation, with the fæces dry and covered with mucus ; and sometimes slight colic. Later on a discharge, which at first is serous and subsequently sero-mucous, issues from the eyes, nose, and vagina ; and also saliva flows from the mouth. The fæces gradually become thinner and thinner until finally violent diarrhœa, accompanied by colic, sets in. The evacuations are then fœtid, viscid, and sometimes mixed with blood. Frequently they are passed with considerable tenesmus, and even with prolapse of the highly congested mucous membrane of the rectum. The animal becomes rapidly emaciated,

staggers when walking, is very sensitive to pressure on the loins, and lies down a great deal. In exceptional cases the patient becomes highly excited, and even mad (congestion of the brain ; the so-called nervous form of rinderpest). Others exhibit violent dyspnœa, and symptoms of severe inflammation of the lungs (coughing, rhonchi, and dulness on percussion ; the so-called pneumonic form).

In the further progress of the disease, characteristic changes are to be seen on the mucous membranes. Red patches which may be flat or in the form of wheals, and quickly become covered with a greyish-white loose crust, appear on the mucous membrane of the lips, tongue, cheeks, gums, nostrils and vagina. The uppermost epithelial layer consequently becomes opaque, and yellowish-grey spots develop on it. Less frequently the crusts are formed from the tumours by their pulpy, caseous disintegration. The crusts on being shed, leave dark-red, hollow places (the so-called erosion ulcers), which readily bleed. In slight cases of the disease there may be no crusts or erosions. We may sometimes notice a similar eruption in the form of very minute pimples and pustules (which form an eschar) on the abdomen, inner surface of the thighs, perineum and udder, in which case it may be confused with that of variola. Pregnant animals frequently abort.

If the disease takes an unfavourable course, the emaciation and loss of strength become more and more increased ; the animal keeps continuously lying down, grinds its teeth, and shivers. Discoloured, purulent, and fœtid discharges flow from the mouth, nose, vagina, and anus. The vagina and anus remain open ; the temperature falls below normal ; and the animal may die in convulsions, or may pass away very quietly. In some cases bloody froth is discharged from the mouth and nose before death.

**Course and Prognosis.**—In unfavourable cases, which are by far the most common, death usually takes place between the fourth and seventh day after the appearance of the first symptoms ; and in particularly severe attacks as early as the fourth or fifth day. In the few favourable cases, recovery takes place slowly with a gradual decline in the intensity of the symptoms, so that the period of convalescence is somewhat prolonged. The progress of the disease in a herd or shed is rather slow and proceeds in successive attacks, but increases in speed as it goes on. At the commencement, only a few neighbouring animals

are attacked and then greater numbers, so that several weeks may pass before the entire herd or all the animals in a shed become infected.

The course of the disease varies greatly according to the character of the epizootic and the breed of cattle. At the first outbreak of an epizootic the disease runs a much severer course than towards the end. The grey cattle \* of the steppes † of Southern Russia, Hungary, Roumania, Moldavia and Servia take rinderpest in a much milder form than other breeds; for they possess, to some extent, hereditary immunity. As they often manifest the symptoms of the disease in a manner which is by no means well marked; it is not always easy to recognise the disease in them. They frequently recover from an attack in 8 days. The mortality in ordinary cattle varies from 90 to 95 per cent.; but in the grey steppe-cattle it is not more than from 30 to 50 per cent.

[As a result of his researches during the years 1899 to 1903 Dr. Lingard, who is the Imperial Bacteriologist of India, states that "In India and the East, rinderpest has been existent probably for hundreds of years, and it is extremely difficult to ascertain in such a vast extent of territory, what immunizing effect may have been acquired by some of the cattle of this country. It is probable, however, that in some districts the cattle have received a certain amount of protection against rinderpest through their progenitors, but up to the present this has been impossible to estimate." We learn from him that the cattle in the plains of India possess a relatively large amount of immunity, but that the Hill (Himalayan) cattle are much more susceptible. He also tells us that the Aden breed of cattle "is most susceptible to rinderpest, probably as much so as Hill animals, on account of the isolated position of Aden, and the infrequency of the visitations of cattle disease to that portion of Arabia."—Tr.]

In *sheep* and *goats* the disease is milder, and its infectiousness is less than in cattle, although the symptoms are essentially the same. Among 4,000 affected sheep in Austria during the years 1859 to 1863, and among the same number in Hungary from 1861 to 1863, there was a mortality of 66 per cent.; and one of 60 per cent. among 30,000 in Russia during 1878. Differing

\* These Russian cattle vary in colour from dark iron grey to a light grey, cream or dirty white. They somewhat resemble the Mysore breed of cattle (the bullocks of which are largely used throughout India for draught purposes), except that they have no hump, and have thick curly hair on their foreheads. They are capital beef producers, but are poor milkers.—Tr.

† A steppe, in the Russian acceptation of the term, is an uncultivated plain, devoid of forest.—Tr.

from cattle, sheep often suffer from pneumonic affections when attacked by rinderpest.

**Rinderpest of Pigs.**—Pluning states that he saw pigs in Sumatra suffering from rinderpest. The symptoms and course of the disease are said to have been the same as in cattle.

**Differential Diagnosis.**—The correct diagnosis of rinderpest is often a matter of considerable difficulty at the first appearance of an outbreak, especially in cases which occur sporadically and in an unexpected manner ; because rinderpest exhibits no symptom that cannot be found in other diseases. Consequently our diagnostic deduction must be made from the combined information obtained from symptoms, autopsy, progress of the epizootic, and history of the particular case under consideration. The chief diagnostic symptoms are : high temperature ; red spots and yellowish-grey coating of the mucous membranes, with subsequent formation of erosion ulcers, especially on the vaginal mucous membrane ; mucous discharge from the mouth, nose, eyes, and vagina ; grave intestinal symptoms ; and excessive emaciation. The proof or the possibility of an infection is of the greatest importance. We should be careful not to confuse rinderpest with diseases which resemble it, especially with the following :—

1. The *malignant catarrhal fever of cattle*, which, however, is only very slightly contagious. It is generally stationary, runs a slower course, and is localised chiefly on the head and respiratory apparatus. Here the severe affection of the eyes must be carefully considered.

2. *Foot and mouth disease* shows a certain resemblance to rinderpest in the ulcerous changes in the oral cavity, in gastric attacks, and in the exanthema on the udder. The eruption on the buccal membrane is, however, very characteristic, and the exanthema is also found on the skin of the digits. The process is usually benign, and the disease spreads rapidly.

3. In *dysentery*, the diarrhoea appears earlier and contains more blood. The intestine only is implicated, and the affections of other mucous membranes (mouth, eyes, nasal cavities, and vagina), are absent. The *post mortem* appearances are also different.

4. *Mycotic inflammation of the intestines*, caused by various fungi, spoilt distillers' wash, etc., may possess great similarity to rinderpest in consequence of its severe course, and, at times, enzootic appearance. Here, also, intestinal troubles and certain

nervous affections are more prominent than in rinderpest. Besides, it is not contagious.

5. *Corrosive agents and scalds*, caused by the partaking of hot distillers' wash, give rise to symptoms of corrosive gastroenteritis, stomatitis, and pharyngitis. The resulting affections are, however, not contagious.

6. *Anthrax*, the gastric form of which may be confused with rinderpest. It runs a much more stormy course, and is not directly infectious. Its presence can be positively proved by *post mortem* examination and by the microscopic demonstration of the bacilli.

7. *Pleuro-pneumonia*, with which rinderpest is sometimes complicated, can in most cases be distinguished with certainty by physical examination of the thoracic cavity.

8. *Rabies*, which might have to be considered in the nervous form of rinderpest, and *cow-pox* are easily recognised.

9. *Infectious hæmaturia* (hæmoglobinuria) of cattle may, according to Babes, be sometimes confused with rinderpest. It is, however, not contagious. Janson states that in Japan the so-called "cattle influenza" may be mistaken for rinderpest.

**Anatomy.**—The pathological-anatomical changes in rinderpest essentially implicate the mucous membrane of the abomasum, small intestine, oral cavity, rectum, and vagina. The general changes which are also common to other infectious diseases are of less importance.

The cadaver is greatly emaciated, the thighs are soiled with fæces, and the neighbourhood of the mouth, nostrils, eyes, anus, and vagina is covered with a yellowish or discoloured mucopurulent discharge. Nodules and pustules are sometimes found on the skin, and especially on the udder. The mucous membrane of the mouth and the pharynx is congested in spots, swollen, covered with lumps of mucus, and exhibits rounded yellowish-grey caseous plates or deposits, which may be seen with the microscope to consist of detritus, cells, nuclei, and micrococci, and are caused by a superficial diphtheritic inflammation. The removal of the plates discloses ulcerous and highly-congested depressions in the mucous membrane; the so-called erosion ulcers.

These changes are best marked on the inner surface of the lips, lower surface of the tongue, buccal mucous membrane, and gums of the inferior maxilla.

In the *first three stomachs* the mucous membrane is congested

in spots, and the epithelium is so loose that it can be easily detached. The contents of the rumen and reticulum are soft, and those of the omasum are often dried up, which is, however, a condition that is also met with in many other diseases. In some cases the contents of the omasum are fluid. Generally the abomasum is empty, and contains only a small quantity of a tough, muco-purulent, yellow, or sanious material. Its mucous membrane is highly congested, especially in the neighbourhood of the pyloric orifice. The congestion is partly diffuse, partly in spots, in the form of points or in streaks; and its colour may be purple, cerise, or reddish-brown, with a tinge of slate-grey. The epithelium exfoliates. On the mucous membrane we may find small, brown-yellow caseous deposits, in the form of plates, which become detached in shreds, and leave behind indented, highly-reddened places, which are studded with blood points (erosions). The glands (peptic and mucous [glands]) of the stomach are swollen, and show considerable cellular hypertrophy:

Exactly the same changes are present in the *small intestine*, in which we find a good deal of swelling and congestion of the mucous membrane, with insulated, scab-like caseous deposits and erosions. In very severe cases these deposits form tube-like casts of the intestinal canal. At the same time there is considerable infiltration of the solitary glands and of Peyer's patches, which are swollen and prominent, and either undergo a purulent disintegration, in which case their contents will be discharged in the form of a plug if pressure be exerted on them, or they become covered with caseous or frequently purulent deposits, which are surrounded by a hyperæmic ring, and which, on becoming detached, leave ulcers behind. Peyer's patches often assume a sieve-like appearance, that is to say, they become areolated. Lieberkühn's glands are considerably swollen, and the intestinal villi cellularly infiltrated. According to Klebs, the entire mucous membrane is infiltrated with micrococci, which accumulate chiefly in the neighbourhood of the vessels, and sometimes occlude the lumen of the vessels.

In the *large intestine* the inflammatory changes are much less pronounced, and are greatest in the cæcum. Generally the mucous membrane is merely swollen, congested in spots, is of the colour of slate or of the skin of an eel, and is covered with mucus. Sometimes the changes in the small intestine may be only of this nature, although in severe cases the entire mucous membrane of the small intestine down to the muscularis may become necrotic.

Frequently the *mesenteric glands* are greatly swollen and infiltrated, so that they resemble in consistency the medullary substance of the brain. In other cases these changes are absent. The *liver* has the appearance of clay, is discoloured, and soft. The *gall-bladder* is filled with thin gall in consequence of stenosis of the orifice of the biliary duct. The mucous membrane of the gall-bladder is swollen, congested, and may even be covered with grey-yellow [buff] plates of exudate. The *kidneys* show parenchymatous changes, are yellow-brown in colour, and abnormally soft. The mucous membrane of the urinary bladder is congested, and covered with mucus. Similar, and usually severer changes are seen in the mucous membrane of the uterus, vagina, and rectum.

The *nasal mucous membrane* is of a dark-red colour, and covered with grey-yellow soft scabs, after the removal of which the true tissue of the mucous membrane lies bare. Similar changes are found in the larynx and trachea, where the deposited masses are frequently purulent and of a creamy consistence. The lungs are sometimes hyperæmic and reddened in spots; sometimes œdematous and even hepatised, or emphysematous. Even pneumothorax and subcutaneous emphysema may be present. The *heart* is flabby, relaxed, and infiltrated with ecchymoses. The endocardium assumes at an early period a dirty purple colour. Hæmorrhages are found under the endocardium and epicardium; and a yellowish transudate in the pericardium. The *blood* is dark in colour, and coagulates with difficulty. There is a large increase in the number of the leucocytes, and the red blood corpuscles show various changes in form (poikilocytosis). Frequently the *nervous central organs* and their membranes are highly hyperæmic. An accumulation of red transudate is sometimes found in the cerebral ventricles and under the arachnoid.

The anatomical changes just described may greatly vary according to the character and the degree of the disease, and age, breed, and state of nutrition of the infected animal. In ordinary cattle the changes are always better marked than in cattle of the steppes, and in sheep.

**Therapeutics and Inoculation of Rinderpest.**—The treatment of rinderpest is quite as unsuccessful as that of many other infective diseases which have a typical course. Besides, it is forbidden by the regulations for epizootics on account of the danger of infection. Hence, affected animals must be imme-



diately killed. For certain districts, as the steppes of Russia, inoculation may, however, be regarded as an important prophylactic agent, though only after an outbreak has occurred. Protective inoculation should never be practised, as it is very liable to spread the disease. The laws of all the European States, with the exception of Russia, forbid inoculation, even after the disease has broken out; because pole-axing is much more effective, and because the mortality (about 36 per cent.) caused by the inoculation of ordinary cattle is extremely high. In the cattle of the steppes it amounts only to about 10 per cent. Favourable results of inoculation are most frequent towards the end of the epizootic.

The technique of the inoculation is very simple. A clean sponge is introduced into one of the nostrils of an affected animal, and is allowed to remain until it is saturated with nasal mucus. It is then removed, and its contents are squeezed out into a small glass, which is closed up. A drop of this inoculation material is then injected subcutaneously on the neck by means of a Sticker's syringe. Even in Russia, where there were four inoculation institutes, inoculation has fallen more or less into disrepute. It is possible that, later on, inoculation of attenuated material will give more favourable results.

[Dr. Lingard has had admirable results with the inoculation of anti-rinderpest serum during the year 1902-1903. In Bengal, the death rate from inoculation with serum was 1.09 per cent. (112 in 10,190); in the Punjab, 0.0019 per cent. (4 in 23,861); in the provinces of Agra and Oudh, 0.015 per cent. (2 in 1,353); and in Burma, 0 per cent. (0 in 4,006). As there was an outbreak of rinderpest in Bengal at the time of the inoculation, "the total deaths of 112 in 10,190 animals injected with serum, instead of being an argument against the utility of the protective serum, is strongly in favour of the good work accomplished by it, for in any outbreak of the disease, whatever moment may be selected for the injection of the animals, a certain number must always have the contagium of the disease incubating in their systems" (*Lingard*). Dr. Lingard points out that the cattle of the Punjab and Burma have a high degree of natural or acquired immunity.

During 1897, Koch and Edington have, more or less successfully, carried out extensive protective inoculation experiments against rinderpest with infected bile. The most hopeful results, however, appear to be obtainable from the method of Danysz, Bordet, and Theiler, a translation of whose *Report on Rinderpest to the Transvaal Government* may be seen in the *Veterinary Record* of the 26th February, 1898, to which number of that periodical I beg to refer my readers for full details. These observers at first confer comparative immunity, by the injection of immune blood, on experimental cattle, to which they then transmit rinderpest by natural infection, with the result that these partially protected cattle

take the disease in a mild form, and, on recovery, become immune for the remainder of their lives. In this, these pathologists act on the knowledge that the injection of blood serum obtained from cattle which have recovered from rinderpest, renders the experimental cattle capable, for a short time, of withstanding the effects of the disease more easily than they would otherwise do ; although it does not prevent them from taking the infection. In this method, defibrinated blood is used in preference to blood serum ; as it is more economical in practice, and is at least equally effective.—TR.]

## CHAPTER II.

## MISCELLANEOUS INFECTIVE DISEASES.

TEXAS FEVER — CARCEAG — INTERMITTENT FEVER—SCARLET FEVER —  
 CHOLERA—FEBRIS RECURRENS—YELLOW FEVER—MEASLES—CADEIRAS  
 DISEASE—BERI BERI—BRADSOT—PROTEOSI—MILK SICKNESS — AFRICAN  
 HORSE PLAGUE—AFRICAN HORSE DEATH—KARASSAN—AKPAIPAK—  
 BLACK DEATH.

**Texas Fever** is a disease of cattle which occurs epizootically in North America, and which in 1894 became known in Germany by the fact of its presence having been proved in American cattle that had been imported into Hamburg during that year. Smith and Kilborne have furnished us with a good deal of exact knowledge concerning its pathology. According to them, Texas fever is an infective hæmoglobinæmia which is caused by the presence of blood parasites in the red corpuscles. These hæmatozoa have received the name of "pyrosoma bigeminum,"\* and are carried by cattle-ticks (ixodes or boophilus bovis) that act as intermediate bearers. The disease occurs in two forms, one acute (midsummer), the other chronic (autumn). The most important symptoms of the disease are fever, hæmoglobinuria, alteration in the form of the red corpuscles, hydræmia, anæmia, loss of appetite, stoppage of rumination, decrease in the secretion of milk, constipation alternating with diarrhœa, dyspnœa, and rapid emaciation. The disease sometimes runs a fatal termination from exhaustion in 4 or 5 days; in other cases, not till 14 days, or even later. Relapses are so frequent that the disease may last for months. The mortality is high, especially in summer; but is less in cases of the autumnal chronic form. The anatomical changes are: the presence of pyrosomæ in the red blood corpuscles; poikilocytosis; anæmia; a thin and watery condition of the blood; hæmoglobinuria; and hyperæmic swelling of the spleen. Besides this, we find ecchymoses in the heart; gelatinous swelling of the connective tissue in the neighbourhood of the kidneys and portal vein; enlargement of the liver; anæmia and biliary engorgement in the brown-yellow liver; and diffuse reddening of the gastro-intestinal mucous membrane.

\* The parasite of Texas fever is termed *Piroplasma bovis* by Drs. Stephens and Christophers (*The Practical Study of Malaria*). It is from 2 to 4  $\mu$  in length, and from 1 to 2  $\mu$  in width.—TR.

The microscopical demonstration of the blood parasites is conducted, according to Kitt, in the following way: Make a preparation of the blood or of the organic juice in the usual way adopted for the staining of bacteria. The drop of blood must be placed as quickly as possible on the cover-glass, and spread out at once into a thin film by means of the edge of a second cover-glass. After allowing the preparation to dry in the air, we draw it through the flame. According to Smith, it is better to expose the cover-glass for 1 to 2 hours to the effect of dry air at a temperature of  $110^{\circ}$  to  $120^{\circ}$  C. After this, the layer of blood is stained with a solution of Löffler's methylene blue either by putting a few drops of the solution on it or by floating the preparation for  $\frac{1}{2}$  to 2 minutes, on this solution in a glass capsule. We can also simply use a watery solution of methylene blue; but this method will take more time. The preparation is then washed in water, dipped for one moment into a 1 per cent. solution of acetic acid, and washed again with distilled water. The cover-glass can then be placed with the adhering drop of water on the object bearer for examination, or it may be mounted in Canada balsam.

[Lingard terms Texas fever *Hæmoglobinuria* or Red Water in cattle and buffaloes, and gives the following synonyms: *Tristeza* (Argentina), *Tick fever* (Australia), *Lal peshab* (Urdu), *Rokto mootra* (Bengali), and *Rodsyge* (Norway). For further remarks on diseases conveyed by ticks, see p. 475 *et seq.*]

**Carceag** of sheep is an epizootic hæmoglobinuria allied to Texas fever. It occurs chiefly in the delta of the Danube, and is caused, according to Babes and Starcovici, by a blood parasite (*hæmatococcus*). It is also supposed to give rise to hæmoglobinuria in cattle in Roumania. The symptoms consist of rigors, fever, colic, hard bloody fæces, anæmia, and in rare cases to hæmaturia. By *post mortem* examination we find the hæmatococci in the red blood corpuscles, hæmoglobinuria, swelling of the spleen, intestinal hæmorrhage, an anæmic condition of the muscles, and a yellow gelatinous œdema in the subcutis.

It is stated that **intermittent fever** (malaria, *febris intermittens*) has been occasionally observed in domestic animals.\* In men it is a pure miasmatic infective disease which occurs everywhere except in the Polar regions, and particularly in swampy regions (Roman Campagna, Pontine Marshes, Sicily, Hungary, the countries of the Lower Danube, and the tropics). Laveran attributes its occurrence to the presence of protozoa (malaria amœbæ). Its chief symptom is an attack of fever which generally lasts for only a short time, and possesses a strikingly regular type (daily, every second, every third, or every fourth day, etc.; *febris quotidiana, tertiana, quartana, quintana*, etc.). The spleen, as a rule, becomes considerably swollen. The fever varies greatly in type, and may take the form of pernicious, intermittent fever, remittent and continuous malarial fever, chronic malarial cachexia, or masked intermittent fever with neuralgia. The chief remedy for malaria is quinine.

\* Lingard states that the majority of horses in the Karnal Remount Dépôt (India) suffer from fever of an intermittent type.—TR.

If we compare the cases of intermittent fever observed in our domestic animals, we must admit the occurrence of malaria in them. The successful experimental transmission of malaria from men to rabbits and dogs strengthen this view. The administration of quinine rendered good service in the treatment of several cases reported from Italy, which is the home of malaria. Dupuy states that in Senegambia, malaria occurs pretty often among horses, either in a highly acute or in a chronic form, which lasts 3 to 5 months. Out of 46 freshly imported horses, 38 died within 3 months. Popow found in a very swampy district 6 horses suffering from malaria, with a temperature varying from  $39^{\circ}$  to  $41.3^{\circ}$  C. According to Sander, intermittent fever occurs not unfrequently in Africa among horses and cattle. Danilowsky found the amœbæ of malaria (*hæmatozoa malaricæ acutæ*) in the blood of birds. These micro-organisms produce acute attacks of fever, which last from 4 to 6 days.

[The following remarks on malarial fever of man have been taken from the Translator's *Veterinary Notes* :

"The '*plasmodium* of Laveran,' which occurs in different forms, gains entrance into the body by means of mosquitoes of the genus *anopheles*, that act as its carriers. This malaria parasite is a protozoon, and not a plasmodium, which term, in this application, is incorrect, though sanctioned by use.

"This parasite, in the form of very minute needle-shaped organisms, is injected into our blood along with the saliva of the mosquito, at the moment this fly bites our skin. Each of these spores (to adopt Professor Lancaster's nomenclature) enters a red corpuscle, undergoes a stage of development (*amœbula*), and breaks up into numerous spherical spores (*enhæmospores*) which enter the blood by the destruction of the red corpuscle. They then invade and destroy other red corpuscles and, by their continued multiplication, infect the whole of the blood. Other stages of development take place, until their final transformation in the blood is attained. They are then in a suitable state to be sucked up by mosquitoes, and after that takes place, they arrive in the stomach of their carrier, undergo changes and migrations, and at last gain access into the mosquito's salivary glands. They are then ready to make a new invasion. We thus see that these organisms are parasites of the mosquito, as well as of man; and that a human being who is suffering from malarial fever, can infect the mosquitoes of a place which had hitherto been free from that disease. In this way, one human being can indirectly infect another human being. Direct infection from one man to another man can be communicated only by inoculation. Mosquitoes have their regular meals, just after sunrise and after sunset, which are consequently the times for specially avoiding these insects.

"The constitutional disturbance is chiefly due to the destruction of the red corpuscles and to toxins elaborated by these parasites (Manson). 'Observations by Koch, confirmed by Stephens and Christophers, have recently shown that in intensely malarial districts, practically all the young children have malaria parasites in their blood. As the native children get older, their blood is progressively less liable to the infection, and in native adults the parasites are rarely found. Immunity from malaria can therefore be acquired' (*Manson*).

"There are several forms of malarial fever, of which intermittent fever (ague) and remittent or continued fever are the most common.

"Dr. Manson points out that the successive generations of the parasites of intermittent fever tend to become simultaneously mature in the blood of their human host, about the same time every day, every second day, or every third day, according to their species. Thus, those which live for 24 hours, produce quotidian (daily) fever; those which exist for 48 hours, tertian fever; and those whose cycle of development is 72 hours, quartan fever. I have not seen similar paroxysms and remissions in the malarial fever of horses.

"The bilious symptoms often seen in cases of malarial fever are chiefly due to the bile of the patient containing an abnormally large amount of *bilirubin*, which is the bile pigment that gives a yellow colour to the skin and other tissues in jaundice. As bilirubin is produced by the decomposition of hæmoglobin, it follows that the more free hæmoglobin there is in the blood, the greater will be the quantity of bilirubin in the bile. In malarial fever, the blood becomes loaded with free hæmoglobin, owing to the destruction of red corpuscles by the malarial parasites.

"The chief breeding places of mosquitoes are shallow pools which are not large enough to contain fish, and which do not dry up between showers. Hence the best way to rid a district of mosquitoes is to drain or fill up these pools, or to destroy the mosquito larvæ by pouring into these pools a sufficient quantity of paraffin oil to cover their surface; and to kill all the mosquitoes within reach. A large number of the mosquitoes which infest houses, can generally be found resting on the walls in the interior of these buildings during the day.

"Colonel A. H. Morris, who is in charge of the Northern Territories of the Gold Coast, reports: 'I caused all holes which might contain puddles, and so become breeding-grounds for the anopheles mosquito, to be filled up. Some hollows in rocks containing about 18 inches of water, were discovered filled with thousands of larvæ. The Hausas' and Carriers' lines were inspected twice a week in order to insure no stagnant water being allowed to remain in old pots or tins. The general result has been an immense reduction in the number of mosquitoes.'

"The great importance of shallow pools for the breeding of mosquito larvæ is due to the fact that such pools do not contain fish, which would devour the larvæ.

"Equine malarial fever is widely distributed throughout South Africa, where it is generally known as 'biliary fever,' on account of the lining membrane of the eyelids assuming a bright yellow colour, and the mucous membrane of the lips becoming tinged with yellow. The mortality is small."]

Formerly **scarlet fever** of men was regarded as a disease which also affected animals. It is evident that this idea arose from petechial fever having been mistaken for scarlet fever. It is strange that this old fallacy should have recently again cropped up by the assertion that scarlet fever of man is produced by the milk of cows suffering from scarlet fever. Klein even states that he has found a *micrococcus scarlatinus* both in the scarlet fever of men and in the milk and suppurating ulcers of cows suffering from scarlet fever, and that he has transmitted the disease to calves by inoculations of cultures of this micrococcus. He also

asserts that he has found this micrococcus in condensed milk, and that he has inoculated it successfully in calves and mice. As might have been expected, these statements of Klein have not been confirmed. We may point out that cows are absolutely immune to human scarlet fever, the correctness of which statement has been proved by Crookshank, M'Call, McFadyean, Axe, Edington, and others. On the other hand, cows' milk, like many other things, may serve in a few cases as an intermediate bearer of the contagium of human scarlet fever. The supposed cases of scarlet fever in horses are more than doubtful.

It is uncertain if animals under the natural course of events suffer from **cholera**. Haffkine asserts that Asiatic cholera has occurred in pigeons and geese simultaneously with the appearance of the disease in men. Ogata states that he has observed in a dog in Tokio a case of Asiatic cholera with all the characteristic symptoms, and that he demonstrated the comma bacillus in it. Pfeiffer and Gruber state that cholera can be produced experimentally in guinea-pigs by the intraperitoneal inoculation of young cultures, but without any intestinal affection. According to Haffkine, an intravenous inoculation of specially prepared cultures of cholera produces in rabbits a disease which is very similar to cholera, and which is accompanied by "rice-water" evacuations from the intestine. Koch succeeded in causing an infection in guinea-pigs by means of cholera comma bacilli after having artificially produced a disturbance in the activity of the abdominal organs and neutralised the hydrochloric acid of the stomach with a solution of soda. Gamaleia states that he has transmitted the cholera bacilli to pigeons.

**Febris recurrens**, the relapsing fever of men, has up to the present been transmitted by inoculation only to monkeys (spirilla in the blood). Steel states that he has observed among horses in India a disease in which he found in the blood motile spirilla.\* The disease was transmitted to monkeys and dogs by inoculation.

**Yellow-fever** is stated to attack horses and dogs in America and Sicily.

[In *The Lancet*, 19th March, 1904, Sir Patrick Manson states: "As regards yellow-fever, although we do not know the germ of the disease, we know how the disease is acquired. The germ is so minute that it passes through the closest filter and seems to be ultra-microscopic, but we know that it exists, and that, like the germ of malaria, it is transferred from one victim to another by a certain kind of mosquito, the common West Indian tiger mosquito (*Stegomyia fasciata*)." Sir W. R. Rynsey (*The Lancet*, 9th August, 1902) tells us that "in Havana, yellow-fever was endemic for a century and a half. During the past year it has been freed from the scourge by killing the mosquitoes in the neighbourhood of each focus of disease as discovered, and by carefully disinfecting every house that had lodged a yellow-fever patient, in order to destroy the mosquitoes that had bitten a sick person."—TR.]

It is not certain that domestic animals suffer from **measles**.

\* Steel mistook an infusorian for a spirillum (see p. 455 *et seq.*).—TR.

Aruch states that he has seen a case in a dog. The supposed cases of measles in pigs were really those of erysipelas, or of some other disease.

**Cadeiras Disease.**—Rebourgeon has described under this name an equine disease which is met with in the equatorial provinces of Brazil. The symptoms are: stiffness in the hind-quarters, petechiæ on the conjunctiva, trismus, increased reflex excitability, etc. On *post mortem* examination the psoas muscles look as if boiled, and the pia mater is seriously infiltrated. The nature of the disease has not yet been investigated.

[Under the name of *mal de cadeiras* (disease of the haunches), Dr. Elmassian describes a disease which occurs among horses in Paraguay, and which is due to an organism similar to the trypanosoma of Surra (p. 456). "The symptoms—remittent fever, œdema, wasting—resemble those of Ngana and Surra. Most characteristic is the paralysis of the hind legs, from which the disease takes its name. It runs a chronic course, 2 to 6 months. In donkeys 6 to 12 months. There is occasionally hæmoglobinuria" (*Stephens and Christophers*).—Tr.]

**Beri-Beri**, which attacks mankind in the Dutch East Indies, is an infective neuritis multiplex peripherica. Eyckmann states that it is found in monkeys and hens with symptoms of paralysis in consequence of degeneration of the peripheral nerves. Lacerda reports that it is also met with in pigs and horses. Glogner attributes the disease to amœbæ which he says he has found in the red blood corpuscles.

**Bradsot\*** (the quick disease) of sheep is an epizootic which occurs in the North (Iceland and Farøe Islands), especially in one-year-old lambs during autumn. It is a highly malignant epizootic disease and generally runs a very acute course with symptoms of colic. It was formerly mistaken for anthrax, but appears according to the recent investigations of Nielsen to be a miasmatic infective gastro-enteritis. It is caused by an ovoid bacterium (the bacillus *gastromycosis ovis*) 2 to 6  $\mu$  long and 1  $\mu$  thick. The disease sometimes appears as a *gastromycosis*; at other times as a general infection.

\* Bradsot, which is an infectious disease, is well known in Norway and Scotland. In North Britain it is termed "braxy." It occurs only during autumn and winter; is peculiar to sheep; and is confined almost entirely to sheep not over 3 years of age. It is almost always fatal, and runs its course in a few hours. The chief symptoms are: great depression; unwillingness to move; desire to remain recumbent; swelling of the hind-quarters; tympanites; and sometimes frothing from the mouth. We find, *post mortem*, a dark purple discoloration of the abomasum from hæmorrhagic infiltration; more or less congestion and degeneration of the abdominal organs; dark colour of the blood; and rapid decomposition of the dead body, accompanied by a considerable evolution of gas. The flesh can be used with safety as human food. For a summary of C. O. Jensen's article on this subject, see *Veterinarian*, September, 1896.

It is probable that ovine anthrax is not unfrequently mistaken for braxy in Scotland.—Tr.



**Proteosi.**—Perroncito states that this is an anthracoid disease which is met with in Sardinia, and which is produced by a microbe (*proteus virulentissimus*).

**Milk sickness.**—Kimmel describes under this name a "milk disease" which occurs in the centre of the United States of America in cattle, sheep, goats and horses, and which lasts 2 to 8 days, with loss of appetite, constipation, excitement, rigors, etc. It is stated that human beings who drink the milk of such infected animals suffer from a similar disease.

**African horse-plague,** which raged in Syria and Egypt in 1876 among horses, mules and asses, is a very malignant disease. It has been described by Villoresi, Apostolides, and others. The symptoms are: high fever, severe oppression of the brain, swelling and orange discoloration of the conjunctiva, petechiæ on the oral mucous membrane, great debility, and rapid death, frequently after only a few hours. The maximum duration is 2 or 3 days. The *post mortem* examination shows the presence of septicæmia with general hæmorrhages and degeneration of the great parenchyma. Plassio described a similar disease under the name of "typhus of horses."

**African horse-death.**—According to Sander, who investigated it in German South-West Africa, this disease in its two forms (Dunpaarden ziekte and Dikkoppaarden ziekte) is identical with anthrax. Theiler investigated both forms in the South African Republic, and described them as different diseases. [Sander's conclusion with respect to the nature of this disease is wrong. See p. 467 *et seq.*—Tr.]

**Karassan.**—This name has been given to a disease which, according to Wediornikoff, occurs among the steppe cattle in Russia, and which is probably identical with anthrax.

**Akpaipak** is a disease which is seen in the steppes of the Kirghiz among cattle and sheep, and which was supposed to be scarlet fever (?) by Petrowski.

The **black death** (the bubonic plague) is stated to pass to domestic animals and even to rats in China and Hong-Kong.

[Bubonic plague is an infective disease which is caused by the bacterium of Kitasato and Yersin. It is common to man and many of the lower animals, including pigeons and even flies. Rats are particularly susceptible. The chief symptoms are the formation of buboes and the occurrence of fever. Bubonic plague is characterised by the fact that its virulence is capable of great modification, according to, apparently, the conditions under which the bacteria are cultivated. Thus its course may be very severe and accompanied by a high mortality in one place, and mild in another locality. The specific micro-organisms, which closely resemble those of chicken cholera (p. 98), are chiefly found in the lymphatic glands and internal organs, and in fewer numbers in the blood. They are also present in the fæces, urine, and saliva.

The contagium is fixed, or at least it can be carried only in the form of dust, or along with dust by the agency of currents of air. The chief gates of infection are the alimentary canal and wounds of the skin and mucous membrane. Persons who live under good sanitary conditions, even if they go freely among plague-stricken patients, enjoy immunity to a very great extent. In the early part of 1904, plague was causing a great number of deaths in the Bombay Presidency (India); and it had also broken out in Johannesburg.

"Yersin, Calmette, and Borrel have shown that intravenous, intra-peritoneal, and subcutaneous injections of gelatine cultures of plague bacillus, mixed with a little bouillon and heated for one hour at 58° C., if employed in doses just short of producing a fatal issue, and repeated 3 or 4 times at intervals of 15 days, render rabbits immune to plague bacillus. They further found that the serum of such an immunised animal injected into an unprotected rabbit exercised both an immunising and a therapeutic influence. They also immunised a horse by intravenous injections of living virulent cultures, and the serum of this animal was both preventive and curative of inoculated plague in rabbits, guinea-pigs, and mice. The next step was to test the value of this serum on man. Recent accounts lead us to infer that this has been done, and with the most signal success" (*The Practitioner*, January, 1897).—TR.]

## CHAPTER III.

### CHRONIC CONSTITUTIONAL DISEASES.

ANÆMIA AND CHLOROSIS—PERNICIOUS ANÆMIA—GENERAL DROPSY—  
LEUCÆMIA AND PSEUDO-LEUCÆMIA—HÆMOPHILIA—SCURVY—GOUT—  
DIABETES MELLITUS—DIABETES INSIPIDUS—OBESITY—SARCOMATOSIS,  
CARCINOMATOSIS, AND SCROFULA.

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#### ANÆMIA AND CHLOROSIS.

General remarks on anæmia—Anæmia of sucking-pigs and young  
pigs—Polyæmiæ (plethora).

**Definition.**—The term anæmia means decrease in the total quantity of the blood and consequent diminution in the number of the red and white corpuscles, and in the amount of the hæmoglobin, albuminous bodies of the plasma, salts, and water. The expression chlorosis signifies decrease only in the quantity of the hæmoglobin; the respective proportions of the blood mass, albuminous bodies, leucocytes and frequently of the red blood corpuscles being often normal. Although in mankind this condition of the blood is common, and affects only females at the time of their sexual development; we have no positive proof that it occurs in our domestic animals. The word “chlorosis” is frequently used as equivalent to “anæmia” by persons who base their definition on the principal symptom instead of on the principal cause. The practice can be accepted only on the understanding that ordinary anæmia of animals is not the same disease as human chlorosis.

**Etiology.**—Anæmia is often congenital, and is chiefly found in puppies, kittens, foals, calves, cattle that are specially bred for early slaughter, too finely-bred pigs, young pigs, and particularly in fancy breeds of dogs. It may appear in fat

animals, and in animals which have lost a considerable quantity of blood at one time or on different occasions. We may mention that in certain districts, animals are bled on several occasions before fattening them. A permanently bad system of feeding, especially on potatoes and food poor in iron, is a prolific cause of this disease (the so-called anæmia of inanition). We often meet with a secondary, symptomatic anæmia after many acute and chronic diseases, exhausting exertion, parturition and abnormal loss of fluid. We may here refer to the remarks made on progressive pernicious anæmia (p. 426). We shall discuss the anæmia of sheep in a separate section (p. 430) on account of its great importance.

**Symptoms.**—Anæmia is recognised by the paleness of the skin and mucous membrane; the colour of the latter appearing as if washed out to almost a milky-white. There is general debility; the patient becomes fatigued by slight exertion; the action of the heart and lungs is accelerated; the pulse small; frequently the temperature is low; the cardiac sounds are sometimes masked by *bruits de diable*; the appetite is poor; and disturbances of digestion appear, with the frequent result, in newly-born animals, of death from exhaustion after a few days. The tone of the muscles becomes lowered, and the limbs show a tendency to swell. The course of anæmia is chronic, and not unfrequently ends in recovery. Pernicious anæmia (p. 426) usually terminates in death. Chronic anæmia of sheep is, as a rule, complicated with hydræmia, which we shall discuss later on.

**Anatomy.**—Besides the deficiency of blood in all the organs, the blood itself in anæmia is pale, it resembles the juice of meat, and shows little or no tendency to coagulate. The heart and the large vessels are sometimes greatly contracted (phyoplasia); and the principal organs, such as the muscular tissue of the heart, liver, and kidneys, frequently undergo fatty degeneration, if excessive anæmia has been present for a considerable time.

**Diagnosis.**—The most important diagnostic appearance of anæmia is the paleness of the visible mucous membranes. We also find by physical examination, deficiency of hæmoglobin in the blood. Zschokke recommends for this purpose Gower's hæmoglobinometer (colour method).

**Therapeutics.**—Next to good, digestible, nitrogenous food, such as milk diet with horses, and a rational system of hygiene, the principal remedy for anæmia is iron. It is best to give it in a form that can be easily absorbed, and in small doses along with the food, or subcutaneously, when the digestion is very weak. The simplest preparation is *ferrum pulveratum*,\* which is prescribed with common salt in gramme doses for horses and cattle, and in decigramme doses for dogs. To smaller animals we give tincture of iron, such as *tinctura ferri pomata*,† in drops diluted with a sufficiency of water. For instance: *tinctura ferri pomatæ* 5; *aqua destillata* 250; M. Of this we may give one teaspoonful twice a day to a dog. Besides iron, arsenic may be exhibited as an alterative. For young animals, especially pigs, we recommend phosphate of lime in the form of soluble, ground-up bones.

**Anæmia of Sucking-pigs and Young Pigs.**—Braasch states that there occurs among young pigs in Schleswig-Holstein a widely-spread anæmia, which yearly causes the death of hundreds of sucking-pigs on some farms, so as to endanger the entire breeding of pigs in that province. The animals are perfectly healthy and fat up to about 14 days after birth, from which time they assume a pale appearance and manifest signs of chlorosis. The anæmia is so great that the young pigs can be castrated without losing any blood. They also show well-marked symptoms of perverted appetite, general debility, dyspnœa, and palpitation of the heart. The animals quickly die if no remedies be applied, or they fall into a chronic state of ill-health, which is accompanied by eczema, cough, diarrhœa, swelling of the glands, etc. The disease is caused by excessive stimulation of the reproductive function of the sows, and by want of variety in their food. As the disease very rarely occurs, except in winter, it seems that the cold to which young pigs are often exposed in badly-managed piggeries (cemented floors without any litter) must have some etiological effect.

**Polyæmia (Plethora)** is the opposite to anæmia. Formerly, plethora was frequently mentioned in text-books of pathology; but now it is rarely alluded to, and even its existence has frequently been denied. Müller and Cohnheim in particular have called attention to the fact that even a 50 to 80 per cent. addition to the blood mass by transfusions of homogeneous blood will not produce plethora. On the other hand, Bollinger pointed to the diversity in the percentage of blood in various kinds of animals. Thus, in a fat pig the percentage of blood as compared with the weight of the body amounted to only 2.2, and in a working horse to 13.5. These extraordinary diversities are arguments in favour of the possibility of plethora, which seems to occur most commonly at the commencement of fattening young, strong animals which have been well fed, but which have had no exercise for a long time; and perhaps

\* Pure powdered iron. It nearly corresponds to *ferrum redactum*.—TR.

† Tincture of malate of iron.—TR.

in cases of sudden stoppage of the secretion of milk. This plethora is characterised by hyperæmia of the mucous membranes, a full and strong pulse and heart-beat, and tendency to congestion of the brain and lungs. In human beings, plethora most frequently occurs from irregularities in living. On *post mortem* examinations in such cases we find hypertrophy and dilatation of the heart, large vessels, and capillaries of the large glands, associated with well-marked hyperæmia.

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### PERNICIOUS ANÆMIA.

Pernicious anæmia of animals—Pernicious anæmia of men.

**Etiology.**—Pernicious anæmia is a particularly malignant, primary or essential, and usually fatal form of anæmia in full-grown animals. Nothing positive is known as to its cause, although certain facts point to its infective or toxic origin. Zschokke having observed in horses several cases of it in one and the same stable, regards it as a stable epizootic. Over-exertion, long-continued standing in the stable, and a previous attack of inflammation of the lungs, may be regarded as predisposing causes. Up to the present time, about a dozen authentic cases have been seen in horses. Imminger states that he has observed the disease as an enzootic among cattle, and Friedberger has described a case in a mule.

Severe anæmia may become secondarily developed as a consequence of illness caused by worms, chronic suppuration, etc. Under this heading we may appropriately place the ovile disease due to distoma hepaticum (liver flukes) and the dochmiasis\* of dogs and cats. The strongylus contortus in sheep, and, in a few cases, the strongylus tetracanthus in foals (after immigration into the cæcum) may also cause pernicious anæmia.

**Anatomy.**—By *post mortem* examination we find, just as in ordinary anæmia, a general bloodless condition of the tissues and a watery state of the blood. The blood shows characteristic changes, which are absent in ordinary anæmia; namely, great paleness of the red blood corpuscles and alteration in

\* A disease due to strongyli in the intestines. In man the parasite is the strongylus duodenalis; in dogs, the dochmius trigonocephalus, which takes up its abode generally in the small intestine and cæcum; and in cats, the dochmius Balsami felis. In all these cases, the presence of the parasite sets up anæmia and various disturbances of the alimentary canal.—Tr.

their form (poikilocytosis). They may be increased in length, may become angular, club-shaped, or biscuit-shaped, or may assume the form of a drum-stick. Also very small (microcytes) and very large (macrocytes) blood corpuscles appear in the blood. On a hæmoglobinometric examination of the blood, the proportion of the hæmoglobin is found to have decreased very considerably. Among other important changes we may mention: fatty degeneration of the vessels, muscular tissue of the heart and skeleton, and cells of the liver and kidneys; extensive hæmorrhages in almost all the internal organs, especially under the serous membrane, in the muscles, large glands of the body, etc; swelling of the liver and spleen; cellular infiltration of the medulla of the bones; and hæmoglobin infarcts in the kidneys. No one particular organ or tissue, with the exception of the blood, is affected to any great extent.

**Symptoms.**—The disease usually commences with symptoms of increasing debility so gradually, that its earliest stage is usually overlooked. In other cases, respiratory catarrh with cough and nasal discharge may be the primary symptom. It also appears that the disease may begin in an acute form with high fever. Sometimes the mucous membranes are at first yellow; but they soon become more and more pale. It is a striking fact that often, even in far advanced stages of the disease, the appetite and state of nutrition continue fairly good. On the other hand, debility, incapacity to bear fatigue, and tendency to sweat may appear at an early period. A characteristic symptom of the disease is the presence of obstinate high temperature, which frequently continues intermittent for days, and which generally defies treatment. This fever is accompanied by a very considerable increase in the frequency of the pulse. The heart sounds are sometimes complicated with secondary murmurs. The blood shows the symptoms, already described, of poikilocytosis, macrocytes and microcytes. During the further course of the disease, the animal gradually loses condition, and sometimes shows signs of dropsy; emaciation increases; and death may take place with great increase of temperature, after the disease has lasted for months, and even for years. Ostertag saw a case of death in a horse with symptoms of paralysis (hydrorachis and hydrocephalus).

**Differential Diagnosis.**—An exact diagnosis can be arrived at only by an examination of the blood, which should

be made immediately after the blood has left its vessels ; because changes similar to those met with in the disease may be produced in the blood by the addition of different fluids (as for instance, distilled water) and by the blood being kept for some time. The absence of a primary affection of any particular organ, excessive anæmia, and presence of persistent intermittent fever, the causes of which cannot be found out, are important points for consideration. In contrast to leucæmia, in which the number of leucocytes is increased, while that of the red corpuscles remains normal, we find in pernicious anæmia no essential increase in the number of the leucocytes, no swelling of the lymph glands, and no leucæmic infarcts. The presence of hæmorrhages is common to both.

**Therapeutics.**—Here, as in ordinary anæmia, the principal object of our treatment should be the regeneration of the blood by preparations of iron. The results, however, are generally very unfavourable. To full-grown horses we give ferrum pulveratum in doses of 2 to 5 grammes in combination with common salt and aromatic powders, or sulphate of iron in similar doses with small quantities of carbonate of potash and aromatic vegetable powders. We may also try arsenic. With horses we commence by giving 10 grammes of liquor arsenicalis, and gradually increase it up to 50 grammes. We may combat the fever by antipyrin, antifebrin, quinine, etc. Unfortunately, however, all these antipyretic agents, even in very large doses, are only too frequently found to be useless. The affected animal should not be worked during treatment.

**Pernicious Anæmia of Men.**—Biermer in 1868 was the first to fully investigate this disease, which consists of an atypical formation of blood, and which by its symptoms and *post mortem* appearances is evidently identical with the pernicious anæmia of the domestic animals. We possess no exact knowledge respecting its origin ; but may hazard the conjecture that it is an infective disease. We must consider as secondary forms, but not as essential pernicious anæmia, those cases in human medicine in which the disease was due to ankylostoma [strongyli duodenalis], found by Perroncio in the workmen in the St. Gothard tunnel, miners, and brickfield labourers, or to bothriocephali. Ponfick made experimental researches respecting the pathogenesis of pernicious anæmia and succeeded in producing it artificially in animals by repeated intravenous injections of blood-dissolving agents (glycerine, pyrogallie acid, and solution of hæmoglobin). Consequently, the essence of the disease seems to be a chronic hæmoglobinæmia in which the described changes of the red blood corpuscles simply signify a separation of the colouring matter of the blood from these corpuscles. This view is strongly supported by the proof of the



presence of infarcts of hæmoglobin in the kidneys, as we have seen in the case of a horse. In the process of chronic hæmoglobinæmia we find interference with the elaboration of the blood, and deficiency of oxygen, with consequent fatty degeneration of the organs of the body and rupture of the vessels. Further research into the cause of the hæmoglobinæmia of pernicious anæmia is desirable. Possibly the disease is due to bacteria or toxins.

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### GENERAL DROPSY (*Hydræmia*).

General remarks—Hydræmia of sheep—Hydræmia of cattle.

**General Remarks.**—There are three forms of hydræmia: (1) A mechanical form caused by engorgement of blood due to heart failure, and to disease of the lungs, kidneys and liver; (2) an inflammatory form; (3) and an hydræmic form caused by a serous condition of the blood, and by alteration of the vessels. The last-mentioned form occurs most frequently in sheep with accompanying symptoms of distomatosis [disease caused by flukes]. Hydræmia develops in pigs and sheep during the course of anæmia without parasitic agency, as will be discussed later on. Apart from this hyperæmia of sheep, the so-called “general œdema of cellular tissue” is another hydræmia of practical clinical importance. It occurs as an independent disease in draught-oxen in sugar factories, and in other cattle, from exclusive feeding on distillers’ wash, and in horses that work in sugar factories. The causes of hydræmia are on the whole the same as those of anæmia; hereditary predisposition and bad feeding being the most important factors. The disease is divided into varieties corresponding to the respective localisation of the accumulated water, as, for instance, anasarca (water in the skin); ascites (water in the abdomen); hydrothorax (water in the chest); and hydro-pericardium (water in the covering membrane of the heart). Anasarca is found at first on the lower parts of the body, as on the legs, hypogastrium, inferior surface of the chest, and scrotum. In sheep, on the contrary, it is seen on the head, which is kept low down while grazing. The skin is swollen and doughy to the touch, and retains the impression of the fingers; but is neither painful nor of abnormally high temperature. Animals affected with hydræmia suffer from debility, shortness of breath, and indigestion; the mucous membranes

are pale and sometimes œdematously swollen; and the pulse is very weak. Besides the watery, thin condition of the blood, we find in all the cavities of the body and under the skin, accumulations of hydræmic fluid, which is clear, colourless, or yellow, free from coagula, and poorer in albumen than the blood or inflammatory lymph. It coagulates only after it has been kept a considerable time. This watery transudate contains a small number of the component parts of cells, especially of endothelial cells, and a few leucocytes.

HYDRÆMIA OF SHEEP (*chlorosis, dropsy, cachexia aquosa*).

**"Rot" in Sheep.**—The term "rot" was formerly applied to any disease which was characterised by emaciation, weakness, poverty of blood, and dropsy. Tuberculosis of cattle was also called "rot" under which heading recent writers have sometimes placed distomatosis [the morbid condition set up by the presence of flukes], and diseases arising respectively from strongyli in the stomach and from tænia; and at other times, only non-parasitic ovine chronic anæmia (chlorosis) and dropsy. As we shall later on discuss the diseases just mentioned, we shall here confine our attention to the non-parasitic constitutional disease which occurs epizootically among sheep, and which manifests itself chiefly by chronic anæmia and general dropsy.

**Etiology.**—The causes of dropsy or chronic anæmia (chlorosis) of sheep, which is a grave disease that attacks whole flocks, may be placed under the following two headings: (1) Insufficient and damaged food, swampy, sandy or boggy pasture land, unsanitary abodes, and bad management; and (2) unfavourable climatic conditions, such as long continued wet and cold weather, badly situated grazing land, and penning the sheep on wet, cold soil. Consequently the disease is more frequent after very bad and wet seasons and inundations, than at other times.

**Anatomy.**—The extraordinary poverty of blood is the first thing that strikes one on viewing the dead body. The blood resembles the juice of meat. The subcutaneous tissue of the neck, abdomen, and lower surface of the chest, and the inter-muscular connective tissue are œdematously infiltrated. The affected connective tissue seems, when it has not entirely disappeared, to be gelatinous; the muscular system is relaxed and pale; the abdominal viscera are shrunken and pale; the lungs are inflated and pale; and the heart is relaxed and flabby. A clear, light yellow transudate is found in the thoracic and abdominal cavities, pericardium, ventricles of the brain, between the cerebral membranes, and in the spinal canal.

**Symptoms.**—The appetite greatly varies, being sometimes good for a long period, and at other times bad from the commencement. The animal is depressed and weak; drags its legs; gives way in the loins; and becomes gradually emaciated. All the mucous membranes are very pale and finally become quite white; the conjunctiva is frequently œdematously swollen; the sclerotic has a blue tinge; and a mucous discharge from the eyes is frequently present. The skin is pale, greasy, and inflated; and the wool is lustreless, brittle, and easily falls out. The frequency of the heart and pulse is increased. (Edematous swellings develop in the intermaxillary space, on the throat, inferior surface of the chest, and hypogastrium. The abdomen increases in size and becomes fluctuating (ascites). Later on, persistent exhausting diarrhœa appears; the animal becomes so weak that it is unable to move, and continually remains recumbent. Usually the patient lingers on for several months, and sometimes for even a year.

**Therapeutics.**—The only remedial means which is of any use is attention to hygiene and to feeding. In the first stage of the disease a cure is always possible if we can put the animal on nutritious food which, like hay and corn, contains a large proportion of nitrogen. A great number of medicines have been recommended, the most effective of which are preparations of iron, common salt, lime and aromatic stomachics. From ancient times, gentian, sweet flag [*acorus calamus*], wormwood, and juniper berries have been given to sheep as stomachics for the object in question. For a flock of 100 sheep we may for instance prescribe the following mixture: *Sodii chloridum* 500 grammes; *gentianæ rad. pulv.*; *calami rhizomat pulv.* *aa* 250 grammes; *ferri sulphas, pulv.* 100 grammes; *M.*, to be made up with ground barley-malt into a mass which the sheep will lick up.

#### HYDRÆMIA OF CATTLE (*Edema of cellular tissue*).

**Etiology.**—Chronic hydræmia of cattle, which is frequently observed in abattoirs as an œdema of cellular tissue, is caused by long-continued feeding with food which contains too much water, such as distillers' wash and beetroot residue. A general hydræmia which occurs in oxen and horses, and less frequently in cows, and which sometimes assumes an enzootic type, is of great practical interest to owners of sugar factories. This disease is chiefly caused by exclusive feeding on beetroot residue,

which, since the introduction of the new method of manufacture, contains only about 5 per cent. of solid matter, with 95 per cent. of water. As the proportion of albuminoids in the solid matter is only about 1 to 10; the beetroot residue contains only about  $\frac{1}{2}$  per cent. of albuminoids. The consumption of such food, combined with hard work, finally brings on hydræmia, which occurs far less frequently in cows; because they are little, if at all, worked, and a large amount of water is removed out of their system along with the milk.

**Anatomy.**—A *post mortem* examination of oxen which have died of general dropsy reveals, in a marked manner, the pale and flaccid condition of the entire muscular system, and the absence of *rigor mortis*. The subcutaneous and inter-muscular connective tissue is infiltrated with serous fluid; the flesh is consequently watery; the cavities of the body are more or less filled with transudate; the viscera are shrivelled up, pale and devoid of fat; and the brain is œdematous. We almost always observe signs of chronic intestinal catarrh.

**Symptoms.**—The disease in oxen of sugar factories becomes gradually developed. The animal shows general debility and paleness of the mucous membranes; becomes thinner and thinner in spite of the possession of an excellent appetite; passes a large amount of urine the colour of water; and the coat is rough and stands on end. Later on, increasingly severe digestive troubles set in, with alternating periods of constipation and fœtid diarrhoea. The patient is salivated and manifests on the limbs dropsical swellings which impede progression and render the gait awkward and clumsy. Œdematous swellings soon appear on the hypogastrium, dewlap, and inferior surface of the chest; and dropsical effusions form in the cavities of the body, particularly in the abdominal cavity, so that the size of the belly is greatly increased. Finally, the animal is unable to keep on its legs; it remains persistently recumbent, and dies from exhaustion, after the disease has lasted from 3 to 6 months.

**Therapeutics.**—The treatment consists essentially in the removal of the causes that produce the disease, which can be cured only, as we have already said, by giving a sufficient quantity of dry food; supposing, of course, that the change in the system of feeding has not been undertaken too late.

Diuretics and aperients have been used symptomatically to combat the dropsical swellings and effusions. Compare here the respective methods of treatment of ascites and hydrothorax.

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#### LEUCÆMIA AND PSEUDO-LEUCÆMIA.

**Nature.**—Leucæmia is a changed condition of the blood in which there is a well-marked and continued increase in the number of the leucocytes. Normally, the proportion which they bear to the red blood corpuscles is about 1 to 350. In leucæmia, however, the proportion rises to 1 to 50, 1 to 20, 1 to 10, and even 1 to 2. We divide leucæmia into three varieties, according to their respective starting points. These varieties may become combined with each other.

1. *The splenic form*, which is due to hyperplasia of the spleen, is said to be distinguished from the other varieties by the presence of large, multinuclear leucocytes.

2. *The lymphatic form* which arises from hyperplasia of the lymph glands of the body.

3. *The myelogenous form* which has its origin in the hyperplastic changes of the red medulla of the bones.

**Occurrence.**—Leucæmia occurs more frequently in our domestic animals than is generally supposed. Up to the present it has been observed chiefly in cattle, horses, dogs, pigs, and cats. It has been also found in mice. Among the 70,000 dogs which were taken to the Berlin clinic in the years 1886–1894, there were 21 cases of leucæmia.

**Etiology.**—The origin of leucæmia in our domestic animals is as little known as it is in man ; although it is generally assumed that injuries, intermittent fever, syphilis and other weakening influences are causes of the disease in human beings. Such supposed causes suggest an infective disease which begins as an infection of the blood-forming organs, namely, the spleen, lymph glands, and red medulla of the bones. The supposition of an infective cause is in no way weakened by the fact that, up to the present, experiments in transmission have given negative results. The observations made in cattle seem to indicate that leucæmia may be developed from chronic catarrh of the

abomasum and uterus. Lucet and others state that they have found specific bacteria in the blood of animals suffering from leucæmia.

**Anatomy.**—Besides the increase in the number of leucocytes, which is the essential condition in this disease, the blood shows a series of other changes. It is very pale (so-called "white blood"), and stains the hands only very slightly red; coagulates very slowly; and secretes between the buffy coat and the blood clots a grey stratum which resembles pus. The spleen sometimes contains almost white, purulent blood. The clots in the heart and large vessels are soft and greasy. They resemble pus, and consist almost entirely of leucocytes. In consequence of the decrease in the number of the red blood corpuscles, the specific gravity of the blood is reduced (from 1.055 to 1.040). The blood, as has been demonstrated in human leucæmic blood, contains several abnormal substances, such as formic acid, acetic acid, lactic acid, uric acid, xanthin, hypoxanthin, leucin, etc. The white blood corpuscles show various pathological changes. Only very few of them retain their normal, granular structure; the remainder being hyaline and transparent (embryonal-typhus). We may also observe nucleated, hæmoglobin-stained leucocytes which are filled with fat granules and which are on the point of decay. Transition-forms between the above-mentioned cells may be met with.

In the splenic form, the spleen frequently becomes enlarged to double or treble its ordinary size, or even more, and becomes 5 to 10 times (even 50 times) heavier than in health. Leisering found in a horse a spleen weighing  $28\frac{1}{2}$  kilos. Hellinger reports the case of a pig the spleen of which weighed 3.7 kilos. We may mention that the normal weight of the spleen of a horse varies from  $\frac{1}{2}$  to  $\frac{3}{4}$  kilo.; and that that of a pig is 150 grammes. The edges of the spleen are blunt; its consistence is firm; and the follicles are swollen sometimes to the size of a pea; although the microscopical structure remains normal (simple hyperplasia).

In the lymphatic variety of the disease, which is often complicated with the splenic form, the lymph glands are enlarged and may swell into tumours of considerable size. Their consistence is generally soft. Frequently the lymph glands of the head, throat, extremities, thorax and abdomen, are hyperplastic; in other cases only a few lymph glands are affected. Peyer's patches and the solitary glands of the intestine are only

slightly swollen, or are normal in size. Swelling caused by congestion sometimes appears in the region of the swollen lymph glands of the extremities, throat, head and eyes.

The medulla of the bones often manifest signs of diffuse hyperplasia and cellular infiltration. In the so-called pyoid form of myelogenous leucæmia, it consists almost exclusively of white blood corpuscles and resembles pus in appearance. In the lymphadenoid form it resembles raspberry-jelly.

Besides the described changes, we find in the various organs, as characteristic of leucæmia, leucæmic infarcts and new growths resembling lymphomata, with more or less enlargement of the affected organs, especially in the liver (Reinländer saw one case in which the leucæmic infiltrated liver of a horse weighed 42 kilos), spleen, kidneys, serous membranes, mucous membranes (abomasum and uterus in cattle), uterus, urinary bladder, ovaries, thyroid glands, subcutaneous connective tissue, lungs, retina, tissues round the vessels, etc. The leucæmic infarcts consist of a diffuse infiltration of the tissue with white blood corpuscles, on account of which, the bronchi and blood-vessels, for instance, are surrounded with a dirty-white covering. The new growths which resemble lymphomata appear as circumscribed tumours of the same histological condition as the lymph glands. In the course of leucæmia we may frequently, though not always, observe hæmorrhages in various organs, especially in the kidneys.

**Symptoms.**—The symptoms of leucæmia are not very distinctive. The first general ones are: weakness, inability to bear fatigue, difficulty in breathing, inclination to perspire from slight exertion, attacks of vertigo, and general paleness of the mucous membranes and skin. The action of the heart is usually accelerated; the pulse is frequent, small and irregular; and auscultation of the heart sometimes reveals anæmic secondary murmurs. In some cases all the lymph glands of the surface of the body become on both sides symmetrically enlarged; in other instances, only certain glands, as for example, those in the region of the pharynx. In horses we may at times notice enlargement of the spleen in the form of a swelling of the left flank. The appetite often continues normal for a long time. In the later stages of the disease it is generally capricious, and gastric disturbances, such as diarrhœa, may become developed. Towards the end, hæmorrhages occur in various mucous membranes, as for instance those of the nose, intestine and bladder; and dropsical swellings from engorgement appear in the region

of the swollen lymph glands. It is characteristic of this disease that, with all these morbid changes, no organic affection is present. Consequently, a true diagnosis can be made only by a microscopic examination of the blood. For this purpose we may obtain blood from a vein by means of a fleam or lancet, or by a simple incision into the skin. It is best to examine the blood with as little delay as possible, and to refrain from adding anything to it.

As a rule, the course of the disease is chronic, and runs on for months or even years. A few acute cases have, however, been described. Owing to the uncertain indications afforded by symptoms in this complaint, the true nature of the disease has often escaped recognition until after death, in cases in which an exact examination of the blood was omitted. Affected cattle often seem to their owners quite healthy up to the appearance of hæmorrhages in the mucous membranes. Prognosis is very unfavourable.

**Differential Diagnosis.**—Microscopical examination of the blood enables us to diagnose with certainty leucæmia from pernicious anæmia and other blood diseases; but not always from diseases in which an increase in the number of the white blood corpuscles may be found, without leucæmia being present, as may be sometimes the case in a state of health, although to a lesser extent, or temporarily. Thus we have cases of leucocytosis (temporary increase of the leucocytes) in pregnancy, directly after feeding and after blood-letting, and during inflammatory diseases in general. A persistent though slight increase is also present in other diseases of the blood, and in glanders, in which, also, hæmorrhages occur in mucous membranes. The essential sign of leucæmia is an invariably large (agreeably to the proportion already stated) increase of the white blood corpuscles. The frequent and sudden hæmorrhages of the mucous membranes sometimes cause laymen to confuse leucæmia with anthrax.

**Therapeutics.**—Treatment is of little use. Besides attending to hygiene, we may give, as in any other affections of the blood, preparations of iron, arsenic (Fowler's solution, 5 to 50 grammes for horses in gradually increasing doses), or quinine, which we have found to be a specific remedy against increase in the number of the leucocytes.

**Pseudo-leucæmia** (*malignant lympho-sarcoma*) is a chronic consti-



tutional disease in which, as in leucæmia, there is a general hyperplasia of the lymph glands of the body, but without increase in the number of the leucocytes. According to our experience, it is by no means rare in dogs. It has been sometimes seen in horses. Nothing positive is known about its relations to other constitutional diseases, or about its origin. Cases of it have, however, been seen to gradually change into true leucæmia. At the beginning of the disease, swelling of the lymph glands is the only morbid change which is apparent, but later on severe anæmia sets in.

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#### HÆMOPHILIA.

**Definition.**—Hæmophilia is a congenital hæmorrhagic diathesis which manifests itself by persistent and grave hæmorrhages from very slight injuries, or by a tendency to spontaneous and copious bleeding, usually in connection with former injuries. We must here distinguish it from the secondary hæmorrhage which is met with in certain septic and toxic diseases (septicæmia and petechial fever), abnormal condition of the blood (leucæmia and pernicious anæmia), and parasitic diseases (dochmiasis of dogs). The traumatic predisposing causes in hæmophilia are, as a rule, superficial wounds of the skin, incisions in operations for fistulæ, dilatation of castration wounds, insertion of a seton or rowel, castration, the ulcers of grease, etc. As far as we know, the horse is the only one of our domestic animals which suffers from this disease. Its origin is unknown. In human beings, several cases of well-marked reduction of the thickness of the intima of the arteries, and stenosis of the large vessels with consequent hyperplasia of the vascular system, have been observed. Some authorities believe that the disease is caused by an abnormal plethora of blood in the vessels. Hereditary tendency, even through several generations, may be frequently seen in human hæmophilia.

**Symptoms.**—As a rule, nothing abnormal can be found in the general health of the animal before the hæmorrhage breaks out; and the disease remains latent for a long time, until an external injury starts it. The blood then rushes persistently, as if from a sponge, out of the wound, no matter how small it may be. In spite of the application of all kinds of means for stopping the bleeding, the hæmorrhage usually lasts for hours, and even days. The blood soon becomes watery and ceases to coagulate; the mucous membranes become anæmic; the frequency of

the pulse increases ; and the animal becomes debilitated and often bleeds to death in front of the attendant veterinary surgeon. More rarely, the hæmorrhage stops after some hours ; but may return at any time. Naturally, the prognosis is always unfavourable. Spontaneous hæmorrhages have not been observed in our domestic animals.

**Therapeutics.**—Although we can very seldom master the hæmorrhage by remedial means, success has in a few cases followed their application. Consequently, we may try, first of all, the effect of compression of the wound by ligatures and plugging. If this does not avail, we may employ styptics, such as liquor ferri perchloridi, tannin, alum, creolin, and even the actual cautery. We might also administer internal styptics, for instance, ergot of rye or extract of hydrastis.

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#### SCURVY.

**General Remarks.**—In human medicine the term scurvy is applied to a hæmorrhagic affection which was formerly very prevalent on board ship, in besieged towns, etc., and even now occurs at times sporadically in prisons and barracks. Leaving aside premonitory general symptoms, such as debility, weakness, anæmia, and rheumatic pains ; scurvy manifests itself partly by hæmorrhages of the skin, partly by cyanosis, swelling and hæmorrhage of the gums, and ulcerous gingivitis or stomatitis. The other principal symptoms are : hæmorrhages under the skin, between the muscles, on the mucous membranes and in the internal organs ; and complications, such as pneumonia, pleuritis, and arthritis. Scurvy is probably an infective disease. Bad conditions of food and dwelling, unfavourable climate, and particularly the exclusive use of salt meat on board ship, with absence of vegetable diet, were formerly considered to be the chief causes of the disease ; but now appear, with our present state of knowledge, to be only predisposing factors. Accordingly, Garrod's theory that scurvy is caused by absence of the salts of potash has been abandoned.

Pigs and dogs are the only members of our domestic animals in which scurvy has undoubtedly been found. Formerly, any hæmorrhage coming from the gums was erroneously called scurvy. Almost all the cases of scurvy in dogs which have been

described in books may be referred to simple ulcerous stomatitis, or perhaps, in a few instances, to septic and toxic affections; those in horses, to petechial fever or septic processes; and those in sheep and goats, to anæmia or rachitis. Very few of these descriptions were made with any approach to scientific exactness. Scurvy is evidently rare in dogs; for we met with only 3 cases of it among 70,000 sick dogs.

**Etiology.**—In pigs the chief predisposing, if not actually exciting, causes of scurvy are unsanitary conditions of food and dwelling, and want of exercise. Nothing positive is known about the true cause of porcine scurvy; although Cornevin, Hess, and others consider it to be produced by swine erysipelas. We venture to advance the supposition that scurvy is probably of infective origin in the pig, and also in the dog, which is more exposed than any other domestic animal to those injurious influences which produce scurvy in man.

**Symptoms.**—The first symptoms are debility and a capricious state of the appetite. The gums assume a violet and, gradually, a discoloured appearance, and bleed from slight provocation. The teeth become loose and finally fall out, and the animal gets salivated and exhales a very foetid odour from the mouth. The bristles fall out and show blood on their roots. In dogs and pigs, purple spots and streaks (ecchymoses and vibices), which may change in pigs into deep ulcers, appear on the skin. Swelling of the articulations are met with in a few cases. Besides the spotted hæmorrhages of the gums, we notice in dogs epistaxis, hæmorrhages into the anterior chamber of the eye, and, on ophthalmoscopic examination, hæmorrhages into the retina of the eye. Hæmorrhages of the stomach and intestines may also occur. The animal finally dies from increasing emaciation and debility, which are generally complicated with diarrhœa, if no early amelioration in the external conditions takes place.

The only essential *post mortem* appearances are hæmorrhages in the skin, subcutis, mucous membranes, brain, and under the serous membranes; and a discoloured condition of the blood, which coagulates with difficulty.

**Therapeutics.**—The most important remedial means is the removal of unsanitary conditions of feeding and dwelling. As in human cases, we may administer bitters, astringents (radix gentianæ, cortex quercus, and quinine), and preparations of

iron. It is also usual to give fruit, acorns, and horse-chestnuts. We have given extract of meat in wine to dogs with apparent success.

**Scurvy of Sheep and Lambs.**—Lowak states that this disease occurs along with distomatosis and dropsy and that it manifests itself by the teeth becoming very loose and liable to fall out, tendency to hæmorrhage and ulceration of the gums, fœtid odour from the mouth, and salivation. Other observers have noted the occurrence of continually spreading caries with, finally, perforation of the palate and loosening of the bones of the face, which consequently exhibit an osteoporotic condition. Gips, who has lately made exhaustive researches into the nature of the disease, considers that it has nothing in common with true scurvy; that it is really stomatitis perniciosa of anæmic animals; and that it is due to the gums being easily vulnerable, and to the penetration of particles of food or bacteria into the alveoli of the teeth. The disease, as a general rule, attacks only stall-fed lambs and generally begins on their return from pasture. It spreads epizootically, particularly among delicate animals of fine-wool breeds. Lambs of ordinary breeds, English sheep which are bred for meat, and older animals are almost entirely free. In spring, at the recommencement of grazing, the disease disappears of itself. It is found most frequently after wet summers and when the animals have been fed on watery food, such as distillers' wash and potatoes. According to Gips, the symptoms are as follows: depression, loss of appetite, frothing from the mouth, congestion and swelling of the gums, and separation of the gums from the maxillary bones, defects in the epithelium, and ulcers, which conditions are first observed in the canine teeth, and subsequently involve the incisors and finally the molar teeth. Later on, the teeth become loose, alveolar-periostitis sets in, particles of food become wedged into the alveoli, and necrosis of the maxillæ with painful swellings of the lower and upper jaw takes place. Prehension of food and mastication are naturally rendered difficult and painful, and consequently the consumption of food is decreased and the general health suffers. With increased anæmia and emaciation the animal dies from exhaustion. After slaughter, we find excessive anæmia, a watery condition and pale colour of the blood, which has but little reddening effect on white paper dipped into it, gelatinous softening of the adipose tissue, general dropsy, and chronic gastro-intestinal catarrh. As regards treatment, Gips recommends, above all things, a diet easy of digestion and rich in proteine, such as oats, good hay, barley, peas, tares, lupines, and as much green food as possible. The stalls should be roomy, dry, well-ventilated, free from dust, and should be kept at a temperature from 10° to 12.5° C. The only medicinal agent to be recommended is iron in the form of sulphate of iron (0.1 gramme *pro* lamb), to be given in the drinking water. Gips states that the administration of phosphate of lime is of no use. The teeth may be locally treated with antiseptic and astringent solutions.



is made apparent by the fact that their excrements normally contain a large quantity of uric acid, the elimination of which may suffer decrease in certain cases. Anyhow, it is possible to produce gout artificially in birds by ligaturing the ureters (Elbstein).

[As free uric acid is never found in the blood of birds, mammals or serpents (Sir Alfred Garrod, Dr. Luff and others), the production of gout by ligaturing the ureters of birds, proves that uric acid is formed in the kidneys. The urinary excrement of birds and serpents is composed of an amorphous quadriurate, but it contains no urea. "I have found urea present in the blood of all the birds I examined, viz., the turkey, goose, duck and fowl" (*Luff*). The urine of mammals contains a fair amount of urea, but very little uric acid. Thus the average percentage of urea in human urine is 1.42; and of uric acid, 0.037 (Bloxam).—Tr.]

**Symptoms.**—In birds the metatarsal, phalangeal, and tarsal articulations are the parts which are most frequently and most severely affected; but the metacarpal, carpal, and elbow joints may also suffer. The implicated articulations show at first a soft, painful, and diffuse swelling, which becomes gradually larger and more circumscribed. Often, especially on the lower portion of the metatarsal articulations, we find nodular yellow tumours, which vary in size from a pea to a hazel-nut, and which are usually hard, but may sometimes be fluctuating. They are hot and painful to the touch, and are frequently surrounded by a red area. The epidermis over the tophi ("chalk stones") becomes greatly thickened and exfoliates off in thick layers. These tumours often burst and discharge a gray-yellow [buff-coloured], crumbling or soap-stone looking material, which consists chiefly of uric acid crystals, ammonium urate and calcium urate. These tumours leave ulcers which have edges that readily bleed and a dirty-white base, and which penetrate to the bones. The ends of the bones that form the joints exfoliate from necrosis or become ankylosed, on account of which the toes often become crooked and considerably thickened. Similar swellings, which give the feeling of concretions of lime when felt with the hand, may sometimes be found in the tendons. The general health is usually disturbed. At first the bird can use the affected limb for standing and walking only with difficulty or not at all, on which account it avoids movement as much as possible. Later on, gradual emaciation, debility, poverty of blood, marked paleness of the corpora cavernosa, and exhausting diarrhœa become manifested; and in severe cases the sufferer finally dies.

In visceral gout, the existence of which can be proved only

*post mortem*, we find calcareous nodules and incrustations chiefly in the kidneys, air-sacs, serous membranes of the cavities of the body, coats of the vessels, pericardium, intestines, liver, spleen, and under the skin. The kidneys show signs of nephritis urica, are often greatly enlarged, and their surface is covered with white dots, as if sprinkled with plaster of Paris. The ureters sometimes contain chalky deposits (Kitt).

Bruckmüller met with a case in a dog of nodes on several of the joints, and chiefly on the epiphyses of the ribs. On chemical examination, these nodes were found to consist of urate of soda.

**Diagnosis.**—Gouty inflammation of the joints may very easily be confused with suppurating and caseous tubercular arthritis, which often occurs in fowls. An exact diagnosis of gout can be obtained only by the microscopical or chemical demonstration of urates. If we examine under the microscope the spherical masses of concrete, we shall find that they consist of a regular “felt work” of very fine needles, and that the harder parts are composed of radiating clusters of crystals. The presence of uric acid is chemically proved by placing the concretions on a porcelain dish or watch-glass, moistening them with a few drops of nitric acid, and evaporating the mixture to dryness by means of moderate heat. If the copper-coloured residue be treated with a drop of liquor ammoniæ, it changes into a magnificent purple-red colour, or into purple-blue by the addition of a drop of liquor potassæ. The course of gouty arthritis is very slow.

**Therapeutics.**—The treatment of gout in poultry is chiefly surgical, and consists in opening the suppurating joints. We may give internally alkalis (a pinch of artificial\* Karlsbad salts daily) in the drinking water. These salts, in combination with a large amount of water, are given in human medicine for the object of dissolving the uric acid.

[The researches of Dr. Luff prove that the mineral matter of certain vegetables, such as Brussels sprouts, French beans, winter cabbage, savoy cabbage, turnip tops, celery, and especially spinach, retards the conversion of sodium quadriurate into the biurate, and increases the solubility of the biurate; and that the mineral matter of meat diminishes the

\* Sodii sulphas, *sic.*, 22; potassii sulphas, 1; sodii chloridum, 9; sodii bicarb., 18. Six grammes of this *sal carolinum factitium* of the German pharmacopœia, dissolved in one litre of water, form a fluid resembling that of the natural spring.

solubility of sodium biurate. Hence, these vegetables and many kinds of fruit are an admirable means for relieving sufferers from gout. Strange to say, artificially prepared ash does not produce any good effect in these cases.—[Tr.]

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## DIABETES MELLITUS.

### **General Remarks on the nature of Diabetes Mellitus.**

—The term “glycosuria,” or “mellituria,” is applied to a condition in which there is a temporary excretion of grape-sugar in the urine; and that of “diabetes mellitus,” to a disease characterised by the continued presence of a large amount of sugar in that fluid. Sugar in very minute quantities also occurs, as it is well known, in normal urine, especially in that of cattle and sheep. Besides, we find under normal conditions a small amount of sugar in the urine of female animals that are bringing up their young, as for instance, in bitches after their milk has dried up, in which case milk sugar (lactosuria), not grape-sugar, is excreted with the urine.

The nature of diabetes mellitus is not fully known. This disease, like albuminuria, is probably only a symptom which is common to several diseases. We may, however, safely say that it is due to some defect in metabolism, in which the sugar, instead of being broken up, is excreted in an unchanged condition from the blood. Consequently, diabetes mellitus is equivalent to glycæmia (increased amount of sugar in the blood). The sugar appears to be derived partly from the hydro-carbons in the food, and partly from increased disintegration of albumen. It seems that the increased sugar-forming activity of the liver plays a great part in the pathogenesis of diabetes. In man, the normal amount of sugar in the blood remains constant at about 0.05 per cent.; but in diabetes mellitus it may rise to 1 per cent.

**Occurrence.**—Various authors have met with true diabetes mellitus in dogs. We have seen 7 cases; and Schindelka and Eichhorn 3 each. Heiss saw 2 cases of it in horses; and Rueff and Dieckerhoff, 1 case each. It seems rarer in other animals. Leblanc found it in a monkey.

**Theories on Diabetes.**—The most important theoretical divisions of diabetes which find acceptance at the present time are as follows:—

1. *Neurogenous diabetes.*—In 1849 Cl. Bernard, as we all know, experi-



mentally produced temporary diabetes in animals by injuring a certain spot at the base of the fourth ventricle of the brain, near the origin of the vagus. Schiff and other physiologists showed that a similar result could be obtained by injuring other parts of the central nervous system, as for instance, the posterior lobe of the vermiform process, by destroying the spinal medulla in the neighbourhood of the origin of the brachial nerves, by dividing the sympathetic, the inferior cervical ganglion, the plexus vertebralis, or the last cervical nerve, by irritating the intercostal nerves, etc. Clinical experience with human beings also shows that sugar is found in the urine after certain injuries to the head, concussion of the brain, and in consequence of pathological changes in the medulla oblongata, pons Varolii, cervical medulla, etc., as for instance, in cerebro-spinal meningitis, cerebral apoplexy, fractures of the skull, epilepsy, and in mental maladies (psychoses). Pharmacologists have proved that glycosuria can be produced by various brain poisons, such as morphia, amyl nitrite, carbonic oxide, ether, curare, etc. From the foregoing facts we may infer that neurogenous diabetes is a functional neurosis of the central nervous system, especially of the vaso-motor and sympathetic system.

2. *Hepatogenous diabetes* appears to arise from an abnormal action of the functions of the liver. Even Cl. Bernard showed in his diabetes experiments that puncture of the diabetic area in the fourth ventricle of the brain produces a simultaneous hyperæmia of the liver. It is supposed that, either in consequence of hepatic hyperæmia, the glycogen in the liver is passed rapidly and in large quantities into the blood, and that it is changed there into sugar; or that no formation of glycogen takes place in the liver on account of the acceleration of the blood stream, and that the sugar derived from the food is passed directly into the blood.

3. *Gastro-enterogenous diabetes*, according to Bouchardat and Cantani, is caused by certain digestive disturbances in the gastro-intestinal canal, owing to the absence of certain ferments in that tube. This theory is founded on the possibility of diet exercising an influence on diabetes.

4. *Pancreatic diabetes* is said to have its origin in changes of the pancreas. According to this theory the pancreatic juice, in the exercise of its normal function, changes the hydro-carbons of the food into products which can be fully oxidised in the blood. When the pancreas becomes affected in such a manner that diabetes is set up, the pancreatic juice is so changed that its action on the hydro-carbons of the food does not proceed beyond the production of grape-sugar, which is excreted with the urine.

5. *Hæmatogenous diabetes*, according to Voit, Mialhe, and others, is caused by decreased absorption of oxygen, or diminished alkalinity of the blood, in consequence of which the oxidation of the grape-sugar becomes lessened.

6. *Myogenous diabetes* is said to have its origin in abnormal conditions of muscular activity. Glycogen, which is essentially a nourishing agent for the muscles, and which is completely oxidised under normal conditions, appears, it is said, in considerable quantities as grape-sugar in the blood and urine, when the activity of the muscles has been disturbed. This theory is founded, among other things, on the fact that diabetes mellitus is favourably influenced by muscular exertion and massage.

7. *Constitutional diabetes*, in contrast with accidental diabetes, is

supposed to spring from hereditary predisposition, which is fairly well marked in men.

8. *Gouty diabetes, syphilitic diabetes, diabetes of obesity, diabetes of food (phlorizin)\* and diabetes from confinement* in dogs and cats, are other forms of diabetes mellitus.

9. Some authors believe that diabetes is produced by more causes than one. According to Stokvis, the morbid process in diabetes consists in the disturbance of several functions; for instance, the formation of sugar in the intestinal canal, the formation of sugar and glycogen in the liver, and the consumption of sugar in the muscles.

**Symptoms.**—1. In dogs the first symptoms of diabetes mellitus are dulness, weakness, inability to bear fatigue, and gradual emaciation. If the disease be not too far advanced, the appetite is usually remarkably voracious and the thirst excessive. The urine is very abundant and is more frequently voided than in health. Its specific gravity is generally increased (1.040 to 1.060). The amount of grape-sugar in the urine varies, and may be as high as 12 per cent., which was the proportion found by Haltenhoff in one case. We have observed in dogs, respectively, 4, 5, 7, and 8 per cent. Wolff met with cases showing from 7 to 8 per cent. In such severe instances, the presence of sugar in the urine may be recognised by the sweet taste of that fluid. A grey cataract (*cataracta diabetica*) may become developed in both eyes, which may finally become completely blind (Wolff, Haltenhoff and Ourselves). Schulz and Strübing found bilateral detachment of the retina in a dog which was suffering from mellituria produced experimentally by feeding on sugar. The animals are often affected with vomiting, cough, diarrhoea, hæmorrhages of the mucous membranes (*Thiernesse*), and ulcers of the cornea. The debility becomes excessive towards the end of the disease.

The course of the disease is always slow and extends over several months. Although recoveries may occur, as we have seen in our own practice, the prognosis is on the whole very unfavourable. Death sometimes takes place very rapidly with stupor (*coma diabeticum*). The only anatomical change which we found in one case, *post mortem*, was well-marked fatty degeneration of the liver, with a clay-coloured appearance of the tissues of the liver, and considerable enlargement of the whole organ. Similar changes were observed by Heiss in two horses. We were able to demonstrate enlargement of the liver in a dog

[\* Phlorizin is a glucoside, the consumption of which causes diabetes. Villaret states that it is found in the bark of the roots of apple trees, pear trees, plum trees, and cherry trees.—Tr.]

by palpation during life. Schindelka and Wolff found enlargement and fatty degeneration of the liver in dogs suffering from diabetes. Hence, the anatomical condition of the liver should be connected with the pathogenesis of diabetes mellitus; due weight being accorded to the negative results.

2. Heiss described in detail cases of diabetes mellitus in two Belgian cart *horses*, which were respectively 10 and 11 years old. Both were in the same stable and in the same team. The disease showed itself by debility, depression, atony of the muscles, and general disturbance of health, which was chiefly manifested by the unthriftiness of the animals' coats. The consumption of water rose from 3 to 5 times the normal quantity, and the appetite also became greater than usual. The increased activity of both these functions lasted until death. The nauseous, sickly-smelling urine gave, by Trommer's test, a large, orange-coloured precipitate of suboxide of copper, and was found to contain on an average 3.75 per cent. of grape-sugar. Both horses developed cataracta diabetica and ulcus corneæ in the fifth week of the disease, and died from excessive emaciation and exhaustion 2 months after they had been taken ill. The most important pathological changes found *post mortem* were a peculiar clay-coloured appearance of the liver and enlargement of that organ.

The case described by Rueff was that of a ten-year-old gelding which had been unsuccessfully treated for five weeks, and which was greatly reduced in condition and strength, though the appetite was good and the consumption of water greater than usual. The urine was abnormally copious, and on being examined by a chemist, showed a specific gravity of 1.052, and contained 5.85 per cent. of sugar. Dieckerhoff described a similar case (0.2 to 0.6 per cent. of sugar in the urine).

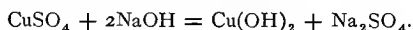
**Diagnosis.**—We may suspect diabetes mellitus when we find that, without apparent cause, the animal becomes gradually debilitated and emaciated, although the appetite continues good or even increased; that the amount of water consumed and urine excreted is increased, and that cataract becomes developed. A positive diagnosis cannot be made without testing the urine for grape-sugar. The easiest test is that of Trommer, in which, after placing some of the urine in a test tube, we add a 15 per cent. solution of caustic potash until a strong alkaline reaction is produced, and then add by drops a solution of sulphate of copper, until the fluid shows a clear deep blue colour (formation

of copper hydrate). If we then heat the test tube, the dark blue colour will gradually change into the opaque yellow or orange of the sub-oxide, which will be precipitated if the urine contains more than 5 per cent. of grape-sugar. The quantitative estimation of sugar with Fehling's solution is based on the same process of reduction.\* Böttger's test depends on the reduction of the sub-nitrate of bismuth into black metallic bismuth by boiling in an alkaline solution; Moore's potash-test gives a deep brown colour to the urine, to which liquor potassæ has been added, after the upper layer of the fluid has been carefully heated. Grape-sugar in the urine can be demonstrated by the polariscope (apparatus of Mitscherlich) as it turns to the right the plane of polarisation; hence the name "dextrose." By this apparatus we may also very easily obtain a quantitative analysis of sugar. A more elaborate method is that of fermentation, in which the yeast causes the sugar to be broken up into carbonic acid and alcohol. Here the loss of weight from the escape of the carbonic acid enables us to calculate the amount of the grape-sugar.

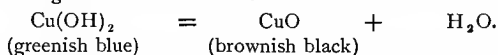
**Therapeutics.**—The treatment of diabetes mellitus chiefly consists in reducing as much as possible the supply of hydro-

\* Mr. W. H. Willcox, B.Sc., A.I.C., has kindly sent me the following explanation of the reactions in these two tests:—

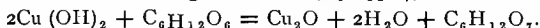
In Trommer's fluid we have in the first instance:



If no sugar be present in this cold solution, the hydrate will form a *greenish blue* precipitate, which will then remain unchanged; but which, on being boiled, will turn brown, owing to the formation of  $\text{CuO}$ . The reaction is as follows:



If, however, sugar be present in sufficient quantity in the cold solution, it will prevent the precipitation of the  $\text{Cu(OH)}_2$ , which will accordingly give a greenish-blue colour to the liquid; and it will, under the action of heat, change the hydrate into  $\text{Cu}_2\text{O}$  (cuprous oxide or suboxide) by the abstraction of oxygen; becoming itself converted into gluconic acid ( $\text{C}_6\text{H}_{12}\text{O}_7$ ). Thus:



Fehling's test is based on the fact that the presence of sodium-potassium tartarate causes the solution of the greenish-blue precipitate  $\text{Cu(OH)}_2$ , which then gives a *deep blue* colour to the liquid. Hence, Fehling's fluid (a mixture of  $\text{CuSO}_4$ ,  $\text{NaOH}$ , and  $\text{NaKC}_4\text{H}_4\text{O}_6$ ) is practically a solution of  $\text{Cu(OH)}_2$ ; and when it is boiled it gives exactly the same reaction as that of Trommer, namely, the formation of the red precipitate of cuprous oxide.

Fehling's test is considered the more elegant one of the two; because it does not entail delay by the precipitation of the hydrate which is held in solution.

Fehling's and Trommer's tests are so delicate that they will easily indicate the presence of a solution of grape sugar of 1 in 5,000.—TR.

carbons in the food. Sugar-formers, the most important of which are starch, sugar itself and glue-yielding substances, should be replaced by albumen and fat. We have proved in two cases that an exclusive meat diet is best for dogs. In human medicine, alkalies, especially Karlsbad water and salt, opium, carbolic acid and salycilate of soda are administered, and saccharine is used so that the food may not be entirely deprived of a sweet-tasting element.

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#### DIABETES INSIPIDUS (*Polyuria*).

**Definition.**—In human medicine the term diabetes insipidus is applied to a disease which is probably due to nervous disturbance, and is characterised by the excretion of an abnormally large quantity of non-saccharine (*insipidus*, *i.e.*, tasteless) and very watery urine. Nothing is known respecting its origin, although it appears probable that it has some connection with diabetes mellitus, as it may change into it. It is stated that diabetes insipidus may be produced in the ordinary course of events by injuries of the brain and various acute morbid processes; and experimentally by injury of the fourth ventricle in the neighbourhood of the centre of the vagus.

It has not been positively proved that animals suffer from a disease which is identical with diabetes insipidus in men. Polyuria differs from diabetes insipidus by the fact that it is merely a symptom, and often only a temporary symptom of morbid conditions produced by the absorption of exuded material during the course and towards the end of many acute diseases, especially contagious equine pleuro-pneumonia, strangles, pharyngitis, diseases of the medulla oblongata and of the cerebellum, many diseases of the kidneys, especially chronic interstitial nephritis (the so-called contracted kidneys), and hyperæmia of the kidneys; and during chronic diseases such as leucæmia, glanders, and tuberculosis. Severe polyuria sets in after the administration of certain poisons, such as cynanchum vincetoxicum, which sometimes gives rise to enzootic poisoning in sheep. The principal cause of the ordinary kind of polyuria, which we often find as an enzootic among horses in badly-managed stables, is, as we all know, the consumption of musty oats. Damp oats having been kept for a considerable

time without being turned over, as for instance on board ship, ferment, probably, from the action of mould fungi. Dammann observed that musty peas also produce this polyuria. This toxic polyuria has just as little in common with true diabetes insipidus as the polyuria of contracted kidneys. The nearest approach in horses to human diabetes insipidus is found in those cases of polyuria which have no apparent cause, and which exhibit no characteristic changes on *post mortem* examination. Stockfleth states that he found true diabetes in a horse after a chill; and Perrin, after a kick on the region of the liver. Schindelka describes, in a dog, one case which was distinguished by its rapid progress, and by complications (cataract and abscess) peculiar to diabetes mellitus.

**Symptoms.**—Although the equine form of polyuria due to musty fodder differs as to cause from human diabetes insipidus, it presents similar symptoms. The digestion becomes impaired a few days after the consumption of the musty oats, and colicky pains may also ensue. Attention is soon attracted to the abnormal frequency of the act of micturition, and to the large quantity of excreted urine, which in severe cases may amount, daily, to 25 and even to 50 litres. The urine is pale, very watery, poor in solid constituents, and of the low specific gravity of from 1.001 to 1.015. It contains neither albumen nor sugar, and often has an acid reaction, probably on account of simultaneous intestinal disturbances. The increased secretion of urine is accompanied by increase of thirst, which often becomes very excessive. Horses have been known to consume in one day from 80 to 100 litres of water. The temperature is usually normal, but may become considerably heightened during severe gastric attacks. Marked debility is generally present.

In the majority of cases the symptoms just described disappear after the supply of the injurious oats has been stopped. It seems that neither recovery nor improvement is possible if the system has been under the influence of the toxic matter for a long time. In such cases the animal becomes more and more emaciated, and finally dies from cachexia after the disease has lasted for months or even for years.

**Therapeutics.**—Removal of the cause, namely, stopping the injurious food, is the best treatment. If this cannot be done the oats may be improved by washing, cleaning,

and frequent shifting.\* It has long been the custom to administer various internal astringents, such as sugar of lead, sulphate of iron, etc. ; but we are unable to express our opinion on their value. Karlsbad salt, opium (as a thirst-quenching agent), valerian root and ergot of rye, are said to be of use in diabetes insipidus of men, and may be tried in chronic cases of equine polyuria.

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### OBESITY.

**Definition.**—Obesity is an abnormally large deposition of fat in the body, especially in the subcutaneous connective tissue. We endeavour to produce it physiologically in animals which are destined for slaughter, and in individuals of certain breeds. Pathologically it occurs chiefly in animals that are kept for breeding, and in dogs, which are often treated for it by veterinary surgeons. Its treatment in animals is of far less pathological importance than in human beings.

**Etiology.**—The principal causes of obesity are : abundant consumption of fat-forming food and want of exercise. Many dogs, especially lap-dogs and house-dogs, and stud animals, such as bulls and boars which remain permanently in their stalls, and which are too highly fed, often become very obese. The adipose tissue is formed from the albumen as well as from the fat contained in the food. The hydro-carbons of the food participate only indirectly in this process, by diminishing the loss of albumen in the body. Hereditary predisposition may be so strong in certain breeds of pigs, cattle, sheep, and possibly of dogs, as to induce obesity independently of the factors just mentioned. Anæmia is another predisposing cause of obesity, as we may learn from the success of the empirical practice of bleeding animals which are intended to be fattened for slaughter.

**Symptoms.**—With increase of the well-known signs of obesity, there is loss of functional power and vivacity. The

\* Heat, applied for instance by kiln-drying, boiling, steaming, or parching (as is done in India), is the best means for destroying the pathogenic fungi. Curtailment of the supply of the horse's drinking water would not only be useless cruelty, but would also have the bad effect of checking the excretion of morbid material from the system.—TR.

appetite becomes capricious, digestion disturbed and the procreative faculty impaired or even destroyed. In cases of long continued and excessive obesity, dyspnoea becomes complicated with acceleration of the pulse, palpitation of the heart, and disturbances of the circulation, such as hypertrophy of the heart, fatty infiltration of the heart, and finally fatty degeneration of the heart.

**Therapeutics.**—Obesity may be treated by giving less food, by reducing the quantity of some of the component parts of the food, or by increasing the decomposition of the adipose tissue.

1. Decrease in the total amount of the food is the simplest and most reliable remedial means. In our clinic we have always succeeded in making fat dogs lean in a comparatively short time by a simple reduction of the diet. This plan is not always practicable; for owners cannot be relied on to enforce the necessary regimen.

2. A decrease or withdrawal of food constituents, such as fat and hydro-carbons, which tend to form adipose tissue, has been tried in man with various modifications, the oldest of which is that of Banting. His "cure" consisted in the almost entire elimination of hydro-carbons and fat, so that the diet was practically restricted to lean meat. Good results may be expected from this method, if it be judiciously applied for moderate periods of time. Its too strict and long continued employment gives rise to aversion from albuminous food, and indigestion, followed by catarrh of the stomach and intestines, general weakness, and exhaustion. On account of the absence from the food of fat and hydro-carbons, which, when digested, protect the albumen of the tissues from disintegration, the body consumes a portion of its own albumen, which, in any case, is not very abundant in obesity. Elbstein's method excludes hydro-carbons, except fat, which may be taken in small quantities. It appears to reduce the feeling of hunger and thirst, and to favour the deposition of albumen in the body.

[In man the only dietetic method for checking obesity which can as a rule be permanently adopted with good results, is the exclusion of starch (bread, biscuits, cakes, pastry, macaroni, vermicelli, rice, sago, cornflour, tapioca, arrowroot, etc.), and a liberal supply of those fruits (apples, pears, grapes, pine-apples, oranges, strawberries, raspberries, plums, peaches, greengages, cherries, etc.) and vegetables (cabbages, cauliflowers, tomatoes, celery, spinach, etc.) which are poor in starch.—Tr.]



3. Fat is best reduced by exercise, which accelerates the circulation, increases metabolism, and strengthens the action of the heart. The benefits of exercise are well shown in the training of horses.

4. Deprivation of water, agreeably to the method introduced by Cœtel, relieves the heart of work by decreasing the amount of blood, and consequently increases metabolism. This method may be utilised in the severe affections of the heart which occur during the later stages of obesity. It can be combined with exercise.

[This system has the very serious disadvantage of tending to check metabolism and to produce constipation. Consequently, its application is often followed by rheumatic affections brought on by undue retention of effete and deleterious material in the system, and by digestive disturbance. —TR.]

5. The old remedies against obesity, such as salts of bromine, alkalies, aperients and diaphoretics, have fallen more or less into disrepute, and have given way to the purely dietetic method of treatment; because they operate too violently, and when successful, they obtain their effect partly by the artificial production of a catarrh of the stomach or intestines. The alkalies which increase metabolism, such as Karlsbad salt, are the least injurious medical agents for the reduction of obesity.

#### SARCOMATOSIS, CARCINOMATOSIS, AND SCROFULA.

THE formation of sarcoma and carcinoma comes into the province of medicine when they occur in the internal organs, such as the stomach, liver, intestines, kidneys, uterus, lungs, brain, etc., or when they spread through the whole body. Sarcomatosis and carcinomatosis often assume in horses, and especially in dogs, the aspect of chronic peritonitis or pleuritis with abundant serous or hæmorrhagic exudations; as extensive new-growths of a sarcomatous or cancerous nature may become localised in the pleuræ and peritoneum. The sarcomata of the abomasum give rise in cattle to chronic indigestion and loss of condition. With the frequent carcinomata of the mammæ of bitches, we sometimes observe metastatic formations in the internal organs, such as the liver, uterus (with a foetid, sanious discharge from the vagina), and lungs (with symptoms of pulmonary con-

sumption). Periosteal generalised sarcomata in dogs may give the appearance of articular rheumatism; sarcomata of the lungs and carcinomata in cattle and horses, of tuberculosis; sarcomata of lymph glands in horses, of glanders or of leucæmia; etc. If such processes last a considerable time, the animals become greatly emaciated. Ulcerations of the new growths are complicated with continued septic fever.

Treatment being out of the question, we can only attempt to strengthen the system by good feeding and to check the increase of the cancer cells by small doses of arsenic in the form of Fowler's solution.

#### SCROFULA.

Scrofula is an historical collective definition, and not an independent disease. According to old clinical text-books, the principal symptoms are swelling of the external and internal lymph glands with suppuration, caseation and induration; catarrh of the mucous membranes; cutaneous eruption; gradual loss of condition; and finally cachexia. The majority of the reputed cases of scrofula are evidently those of tuberculosis, a fact to which Spinola called attention even in his time. Other cases were those of rachitis (principally in pigs), swine fever, parasites in the lungs (*strongylus paradoxus* in pigs), pyæmic polyarthritis in foals, and metastatic strangles (strangles was formerly called "*scrofula equorum*"). In aged animals it was probably mistaken for cases of leucæmia, pernicious anæmia, and chronic intestinal catarrh. Cachectic diseases of an unknown kind were generally classed as scrofula.

## CHAPTER IV.

## ADDENDA TO INFECTIVE DISEASES.

By M. H. HAYES, F.R.C.V.S.

SURRA—TSETSE-FLY DISEASE—SOUTH AFRICAN HORSE-SICKNESS—DISEASES  
CONVEYED BY TICKS—ULCERATIVE LYMPHANGITIS—NOTES ON EPI-  
ZOOTIC LYMPHANGITIS.

## SURRA.

General remarks on surra—Surra in the horse—Surra in the dog—Surra in rats—  
Surra in cattle.

**Bibliography.**—The chief publications on surra are : *Report on Surra disease in the Dera Ismail Khan District*, 13th November, 1880, by Inspecting Veterinary-Surgeon G. Evans, M.D. ; *Report on an obscure and fatal Disease among Transport Mules in British Burma*, 1885, by J. H. Steel, V.S., A.V.D. ; *Bacteriology*, by E. Crookshank, M.B. ; *Report on Horse Surra*, 1893, by Alfred Lingard, M.B. ; *Summary of further Report on Surra*, 1894, by Lingard ; *Annual Report of the Imperial Bacteriologist for 1895-1903*, by Lingard ; and *Report of an outbreak of Surra and Filariasis at Kurnal Depot*, 1896, by Lingard. In compiling the following notes on surra, I have made use, chiefly, of the exhaustive researches of Lingard, who has been engaged in the investigation of this disease for several years in India.

**Definition.**—Surra is a continuous fever which is characterised by alternate paroxysms and intermissions, and is caused by a specific hæmatozoon (*Trypanosoma Evansi*).

**History and Distribution.**—This disease, under the name of "surra" (*Hind.* rotten), appears to have been known

for many years to the natives of the low-lying country on both sides of the Indus on the north-west frontier of India. Haig appears to have observed it in Persia during 1876. Evans found several cases of it in the Dera Ismail Khan country, and was the first, in 1880, to describe it, and to attribute its causation to an animal parasite which he discovered in the blood. In 1885 Steel met with a disease among mules in Burma which he regarded as identical with Evans's surra, and which he believed to be relapsing fever. Gunn observed an outbreak of surra among grass-cutters' ponies at Kohat in 1886; and Nuttall, one among the horses of the 19th Bengal Lancers at Meerut in 1886-87. In 1888 there was an outbreak among the Bombay Tramway Company horses. Since then, surra has become enzootic in Bombay. As regards the question of economic loss, we have Lingard's statement that thousands of horses, ponies, camels, and asses died from it in the Gujerat, Ahmedabad, and Baroda districts during the rains of 1893 and 1894; and that the ravages of this disease in the Punjab and North-West Provinces during 1895 were "truly appalling." Burma has also suffered greatly. "The distribution of this malady seems to be entirely influenced by the physical aspect of the country; being far more prevalent in those parts where floods and inundations occur than in the higher and drier portions" (Pease). If we accept the identity of surra with the Tsetse-fly disease (see p. 464), we must accord to surra a wide distribution in Central Africa.

**Occurrence and Prognosis.**—Surra attacks horses, donkeys, camels, goats, and dogs, to all of which animals it proves invariably fatal, except under special medicinal treatment (see p. 461). Its experimental transmission to rabbits and guinea-pigs also gives fatal results. It occurs among elephants; but statistics are wanting as to its absolutely lethal effect on these pachyderms. Cattle may on rare occasions be attacked; but even then it runs in them a brief and benign course. Man appears to be immune. It occurs among ordinary rats, field-rats (*Nesokia providens*), and bandicoots; but seldom in a grave form. The musk-rat (*Sorex caeruleus*) and mice are immune.

**Etiology.**—There is extremely little doubt that surra is due to the presence in the blood of a flagellated infusorian (*Trypanosoma Evansi*) which breeds successive generations

in that fluid, and which measures from 20 to 50  $\mu$  in length, and from 1 to 1.5  $\mu$  in breadth at its widest part (Lingard). The only reason why this view is not unreservedly accepted as a scientific truth, is that the disease, owing to the difficulty of isolating the infusorian, has not been communicated to healthy animals by inoculation from pure cultures of that parasite. This organism, however, is invariably found, during paroxysms of the disease, in the blood of animals which have acquired surra either naturally or experimentally. Although blood containing these infusoria readily communicates the disease to susceptible animals, it entirely loses its virulence when it is filtered through porcelain, so as to free the liquor sanguinis from the parasite. Also, this organism can be passed through a series of healthy susceptible animals even of different species, with the production of the disease and the demonstration of the parasite in each of them. We have the further proof of the infusorian being the exciting cause of surra, from the "undoubted influence which the presence or absence of the trypanosoma has upon the course of the disease, dividing it into paroxysms and intermissions" (Lingard).

During a paroxysm, we can see under the microscope these infusoria in vast numbers (1 to 350,000 in each c.c.) traversing with great activity every drop of blood examined. When this acute stage has passed, these roving organisms disappear for the time being; the temperature falls; the severity of the symptoms more or less abates; and there is an intermission, during which, at the beginning of the attack, the patient may enjoy apparent health. Although the blood during an intermission may appear under the microscope to be absolutely free from these parasites, its inoculation in susceptible animals will, as a rule, reproduce the disease. The blood of surra-affected horses loses its power of transmitting the disease by inoculation in about 18 hours after death.

Under the microscope, the presence of these parasites in a drop of blood is indicated by an irregularly intermittent and characteristic quivering of some of the red corpuscles, which become much altered in form, although the leucocytes remain unchanged in appearance. After a further and careful examination of this "slightly quivering blood, we may at length see a minute thread-like organism, with eel-like movements, emerge from the mass of corpuscles and move slowly forward, or we may see the little being tugging with all its might at a red corpuscle, endeavouring to detach it from its rouleau" (Steel).

The question of the manner in which these parasites interfere with the health of the affected animal has not yet been settled. When they are outside the animal body and in a dry state, they are killed or rendered inert by prolonged atmospheric heat.

**Pathogenesis.**—Surra is an ectogenous and also an endogenous disease. Its contagium is fixed and can be conveyed only by inoculation or ingestion. Hence, in the ordinary acceptation of these terms, it is neither contagious nor infectious. Stagnant water and grass growing on lately inundated land form favourable resting-places for this organism. It is probable that flies (see remarks on the Tsetse-fly, p. 464) act as carriers of surra from infected animals to sound ones, especially if the latter have open wounds. Surra appears to be a purely enzootic disease. Lingard states that there are no records of its becoming enzootic in a country by its having been imported by surra-stricken animals. Although the disease may appear any time during the year, it usually commences its invasion soon after the rains, and attains its greatest ascendancy from September to December, both months inclusive.

**Progress.**—The clinical aspect of surra is essentially one of progressive anæmia, accompanied by paroxysms and intermissions, during both of which there is a gradual decrease in the number of the red corpuscles and in the amount of hæmoglobin in the blood, with consequent anæmia of the visible mucous membranes.

#### SURRA IN THE HORSE.

From an economical point of view, surra is essentially an equine disease.

**Symptoms in the Horse.**—"The chief symptoms in addition to the fever are the occasional appearance of an urticarial eruption, general or localised, closely following the first rise in temperature, but which may make its appearance at any time during the course of the disease; then the presence of petechiæ on the mucous membranes, chiefly that covering the membrana nictitans; lachrymation; and the exudation of a yellow semi-gelatinous material into the subcutaneous and other connective tissues. There is a rapid wasting and great weakness, although in the great majority of cases the appetite

remains good throughout, no matter how high the fever. There is extreme pallor of the visible mucous membranes, and this is followed at a later period by yellowness. From first to last there is progressive anæmia; the blood at first presents a normal character, but after a varying period of time undergoes marked changes. The white corpuscles are increased in number and the red corpuscles usually cease to form normal rouleaux, lose their individuality, and run together, forming irregular masses, which are at first dark, but gradually, as the disease advances, almost entirely lose their colouring matter and become pale" (*Lingard*).

The respective duration of the paroxysms and intermissions is very irregular. *Lingard* puts it down at from 1 to 6 days. He states that in a few experimental horses the paroxysms lasted from 18 to 22 days.

**Anatomy.**—As a rule, there is, *post mortem*, great emaciation, enlargement of the liver and spleen, petechiæ on various internal organs, and a yellow or an amber-coloured jelly-like exudation in the connective tissue of the throat, chest, and abdomen, about the muscles and other tissues, and especially round the base of the heart. The lungs often show signs of inflammation. The mucous membranes and other tissues are frequently tinged yellow by the colouring matter of the bile.

*Steel* noticed, *post mortem*, ulceration of the stomach which was not brought on by the administration of drugs or irritating food, in about two-thirds of his cases among mules in Burma. In India, this ulceration has not been observed among horses as a sequence of surra.

**Manner and Time of Invasion.**—*Lingard* considers that surra is conveyed to horses in water and herbage, and in grain containing the fæces of rats and bandicoots affected with the disease.

**The Period of Incubation** seems to be liable to great variations. It may be put at 6 or 8 days after inoculation or ingestion of blood taken from an animal suffering from surra. It appears from *Lingard's* investigations that the period of latency may be prolonged to 13 days, if the blood used for inoculation has been taken from a dead animal. When the parasites have been given in water by the mouth, symptoms of surra may not appear for even 75 days. We have no

exact data for determining the time required for the disease to become manifest from drinking, under natural conditions, surra-contaminated water.

**Duration of the Disease.**—Gunn states that the average duration of the disease is about 52 days.

**Prognosis.**—Surra is invariably fatal to horses, unless when treated with arsenic according to Lingard's method.

**Immunity.**—Lingard has proved that one attack of surra does not protect a horse from a second attack.

**Differential Diagnosis.**—Surra has often been confounded with kumree (paralysis of the loins); although the weakness from the general exhaustion of the one is entirely different from that due to the local paralysis of the other. The chronic course of surra and the manner in which the internal temperature varies serve to distinguish it from anthrax.

**Prophylaxis.**—The best way to prevent the occurrence of surra among horses in countries in which it exists is : (1) to see that their water supply is pure ; (2) to avoid giving them grass or hay taken from marshy or inundated ground ; and (3) to exclude the excrements of rats, bandicoots, and mice from the grain, which, if this precaution cannot be carried out, should be parched. I may point out in passing that the fact of mice being immune to the effects of this disease is no proof that their fæces might not contain the surra parasite. Besides, it would not be always possible to distinguish their dung from that of susceptible animals. There is some reason to suppose that exposure to cold and draughts predisposes a horse to surra. During an outbreak of this disease in the Parel stables of the Bombay Tramway Company during November and December, 1888, the horses were exposed to cold night winds, and 10 out of 174 died from surra. When means were taken to exclude these chilling winds, no fresh cases took place.

“ The question with regard to the administration of arsenic to animals at the commencement of the rains, as a preventive, in districts where surra is epizootic, is one worthy of trial. A dose of 5 grains of arsenic in the form of liquor arsenicalis (10 drachms), given once a day in the drinking water to each animal, could not fail to produce a beneficial effect, and could with safety



be continued for a month at a time. The dose of arsenic should then be gradually reduced every three days by half a grain at a time until it is discontinued altogether. An interval of seven days should then be allowed to elapse, and again the same process persevered in with intervals, until the termination of the rains, or end of October" (*Lingard*).

**Treatment.**—*Lingard* having made very elaborate experiments with many different drugs (quinine, the double iodide of mercury and potassium, santonin, mercuric perchloride, carbolic acid, iodine, bichromate of potassium, etc.), proved that the best medicinal agent for surra is arsenic, which has a well-marked effect in diminishing the number of the surra-organisms in the blood of affected animals. Great credit is due to him for having devised the following method of treatment by which horses suffering from this hitherto invariably fatal disease have recovered :—

" 1. Only animals in good or in fairly good condition are suitable to undergo treatment for surra. It will be found impracticable to attempt to cure ill-preserved animals, such as Tonga\* ponies, towards the end of a heavy and hard-worked season.

" 2. It is of the utmost importance for the successful treatment of surra that the disease be recognised, and the animal subjected to the drug with the least possible delay.

" 3. The weight of the animal should be ascertained frequently when practicable, so that the maximum dose of arsenic to be administered during the treatment may be regulated.

" 4. Arsenic should be given in the form of liquor arsenicalis.

" 5. In most cases of surra in equines, a dose of 5 grains may be commenced with, and given twice daily for 48 hours, the quantity being increased by half a grain after every 4 doses have been administered, until 7 grains are reached. The latter amount should be continued twice daily for 7 days in the case of an animal below 800 lbs. in weight; but in Australians 1,000 lbs. in weight and upwards, the dose may be increased by the addition of half a grain up to 9 or even 10 grains, twice daily for the same period. The dose should then be gradually reduced by half a grain or one grain according to the condition of the patient, until a four-grain dose has been reached. Again, if possible, after a period of two days, repeat the treatment, increasing and decreasing the dose of arsenic as above described. But the fact must never be lost sight of, that if arsenic be administered to an animal for a prolonged period, symptoms of gastric irritation will supervene sooner or later. It has been found that by giving kaunji [water in which rice has been boiled] to the animal, morning and evening directly after the medicine, the above-mentioned symptoms have not made their appearance; consequently it will be advisable to give kaunji to the animals from the commencement of the arsenical treatment.

\* A form of curricule used in the Bombay Presidency for posting, etc.

" 6. Daily microscopic observation of the blood should be made, and the number of organisms recorded on a chart for ready reference, together with the temperature, pulse, and respirations of the patient, which should be taken three times daily, viz. 8 A.M., 1 P.M., and 5 P.M.

" 7. Most animals will take the drug in one or two pints of water from a bucket, without any trouble. If this be found occasionally impracticable, the arsenic can be mixed with the corn and bran in the form of liquor arsenicalis; but in obstinate cases the medicine will have to be administered to the animal in the form of a bolus or in a draught.

" 8. Acute desquamative nephritis is a common complication of surra, but it must not contra-indicate the administration of arsenic, although the maximum doses cannot be maintained, otherwise the animal will have a relapse, and ultimately succumb to the disease.

" 9. Animals suffering from surra, as a general rule, have voracious appetites. It has been found by practical experience that the following amounts of hay, corn, etc., are necessary for an Australian horse weighing 1,000 lbs. and over; for on this the animal keeps in fair condition and during convalescence puts on flesh rapidly:—

Hay, 15 to 20 lbs.	} Daily.
Lucerne (fresh), 10 lbs.	
Barley, 8 to 14 lbs.	
or	
Gram [an Indian pea], 6 to 10 lbs.	
Bran, 1½ to 3 lbs.	}
Salt, 1½ oz.	

" 10. When once the organism of surra has vanished for some days from the circulation of the animal under treatment, gentle exercise, commencing with five hundred yards, and gradually increasing to one mile, morning and evening, may be allowed. But if the temperature becomes elevated, all exercise must be stopped, until the former again registers within normal limits.

" 11. The exudation of semi-solid material (œdema) into the subcutaneous tissues, notably that of the lower extremities, sheath and under surface of the abdomen, generally becomes absorbed after a time under the influence of arsenic and exercise, but this may be accelerated by daily 'massage' of the body and limbs.

" 12. Surra horses, under treatment with arsenic for long periods, and which eventually die, at their autopsies frequently present some atrophy of the spleen. Consequently subcutaneous injections of infusion of spleens have been made in order to ascertain whether the material injected would perform the function of the atrophied organ. Encouraging results have been obtained.

" 13. Certain of the horse surra cases under treatment somewhat suddenly developed symptoms of chronic arsenical poisoning. These, which include partial loss of power over the hind-quarters, crossing of the hind limbs when walking, and general unsteadiness, and nervous tremors, usually proved fatal within forty-eight hours, although the further administration of the drug was immediately discontinued on their recognition. In some of the later cases under our treatment, these fatal symptoms have been successfully combated by the subcutaneous injection of infusions of spleen and testicle (sheep)" (*Lingard*).

## SURRA IN THE DOG.

Since 1891, several cases of surra have been observed among imported fox-hounds and fox-terriers in the presidencies of Bombay (Bombay and Bandora) and Madras (Ootacamund). On examination, in some of the cases, the surra infusorian was found in the blood during the paroxysms. The symptoms were, more or less, as follows: "Paroxysmal fever, anorexia, at a later date swelling of the integument about the head and throat, injection of the conjunctival membranes, increased lachrymation, effusion into the joints in some cases, and marked œdema of the limbs and under surface of the abdomen, extravasations of blood into one or both anterior chambers of the eyes, followed by opacity of the cornea, and later on total blindness" (Lingard). It seems not unlikely that foxes, jackals, and hyænas in India sometimes suffer from surra; and that, when caught and worried by hounds, they are capable of transmitting the contagium to their canine pursuers. Lingard calls attention to the instructive fact that surra occurs among hounds during the hunting months (January to the end of March); but among the majority of other animals, from June to December. Surra appears, in the large majority of cases, if not always, to be fatal to dogs.

## SURRA IN RATS.

Lewis (Calcutta and Simla) found this or a similar organism in the blood of rats (*mus decumanus* and *mus rufescens*); Vandyke Carter, in that of *mus decumanus* and *mus rattus*; Crookshank, in that of London sewer-rats; and Wittich and Kock, in that of the German marmot (*crictus frumentarius*); Lingard (Bombay Presidency) has demonstrated the surra infusorian in the blood of rats and bandicoots (*mus giganteus* of Hardwicke). "The blood of rats from certain localities was almost always free from the hæmatozoon, while that of the majority of rats from other localities was affected. The hæmatozoon of the rats appears in and disappears from the circulation at irregular intervals, thus producing paroxysms and intermissions, as in the case of surra in the horse. The presence, in varying numbers, of the hæmatozoon in the circulation of rats does not appear to produce any noticeable symptoms in the great majority of them. But some few rats whose blood swarmed with the hæmatozoon have succumbed to leucocytosis, with extravasations of blood, containing the organism, into the anterior chamber and vitreous humour of the eye, opacity of the cornea, enlarged spleen, and

extravasations of blood, or in some cases ulceration of the mucous membrane of the stomach " (*Lingard*).

#### SURRA IN CATTLE.

As already mentioned (p. 456), surra in India is a rare and benign disease among cattle. " Although during the paroxysms of the disease the blood of the bovine species teems with the hæmatozoon, and their bodies become extremely emaciated, nevertheless ultimately they recover and in time become fat and sleek again " (*Lingard*).

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#### TSETSE-FLY DISEASE OR TRYPANOSOMATOSIS (*Nagana*).

The so-called true Tsetse-fly (*Glossina morsitans*) is the carrier of a hæmatozoon (*Trypanosoma brucei*) which produces this disease. It belongs to the diptera, and is somewhat larger than the true house-fly. " The brownish wings lie closed flat over one another down the back, like the blades of a pair of scissors, while the proboscis (*i.e.* the proboscis ensheathed in the palpi) projects horizontally in front of the head. As pointed out by Col. Bruce, the closed wings thus give the fly 'an elongated appearance.' Measured from the tip of the proboscis to the end of the closed wings, the length of the *Glossina morsitans* is about half an inch " (*Austen*).

Tsetse-flies are confined to Africa, and have been found in various districts on the eastern coast, from Zululand to Somaliland, inclusive; on the western coast, from the Congo river to the Senegal river; south of lake Chad; and elsewhere. " As a general rule it may be said that the Tsetse is confined to damp, hot, low-lying localities, either on the borders of rivers or lakes, or at any rate not far from water. Cover in the shape of more or less thick bush or forest is essential, and the fly is not found on open grass plains " (*Austen*).

There are various kinds of Tsetse-flies, all of which have a prominent proboscis, and both sexes are blood-suckers. Owing to their blood-sucking habits, Tsetse-flies greatly torment domestic animals which are in their haunts. " The fly makes a loud buzzing sound when flying, but after its feed and at rest, it emits a peculiar sharp, shrill note " (*Bruce*).

" During the hottest hours of the day they are most

aggressive, but they bite at any time, even at night, when it is a bright moonlight. Their flight is powerful and noisy; the buzz is full-toned and somewhat highly pitched—not dull and droning like the buzz of the ordinary horse-fly; they do not settle as slowly as the horse-fly, but land with a bump, standing well up on their legs. . . . He inserts his proboscis, lowers his head, and raises his abdomen until it is almost vertical. When doing this, and for some little time after he has commenced sucking, he works his wings, buzzing in a minor key, rather like a bee when held forcibly, though not so powerfully. When the keenness of his appetite has been somewhat appeased, he stops working his wings and sucks in silence. If left to himself, he will suck until his originally skinny barred abdomen becomes a large crimson bead. He is then almost helpless; if touched he will not fly, and if brushed off, he will only go a yard or two, to settle heavily on a bush, or gradually sink down to the ground” (*Crawshay*).

“It is astonishing with what rapidity the flies fill themselves; in as small a space of time as twenty or thirty seconds a fly will become swollen out like a balloon with bright red blood” (*Bruce*).

The blood-parasite of this disease lives in the blood of many kinds of wild animals in Africa, apparently without injuring them in any way, but when the Tsetse-fly takes it up in its proboscis and injects it into one of the domestic animals, death soon follows as a rule. Usually the bite of the Tsetse is harmless to man, although trypanosomatosi in man has lately been observed in Africa.

Theiler states that the Tsetse-fly is dependent for its existence on big game, in the blood of which, Bruce frequently found trypanosomata; and that the more big game are pressed back by the advance of white men, the more the disease disappears.

The Tsetse-fly shows great dislike to the smell of excrement, which, on that account, has often been used as a preventive, by smearing it over animals which have to pass through a Tsetse-fly district. As this fly makes its attacks chiefly during mid-day, it is best to travel with horses or cattle by night.

The points of attack which it seems to prefer are the lower part of the belly, insides of the thighs, and under the tail. Bruce, who investigated nagana (*Zulu*, depressed in spirits) in its native habitat, considers it to be surra; because the organisms of both appear to be almost if not quite iden-

tical, and the principal symptoms of both diseases are the same. Lingard found that the microscopic appearance of the flagellated infusorian of the African malady corresponds exactly with that of surra. Bruce proved that the "fly" is in no way virulent of itself, but that it acts as a carrier of the contagium, after it has fed on the live or dead body of a nagana-infected animal. Nagana is fatal to all members and hybrids of the horse family, except zebras which have been bred and reared in Africa. It is possible that absence from that continent would cause zebras to lose this immunity. Theiler (*Vet. Record*, 1st March, 1902) tells us that the dog is the most susceptible animal and the one in which the illness runs the quickest course. Raillet states that *post mortem* examinations have revealed nothing abnormal in the internal organs of those animals which have died from the "fly." Bruce, on the contrary, describes *post mortem* appearances somewhat similar to those of surra. This observer, following the procedure of Lingard, found that arsenic (given in the food to the extent of 12 grains daily in the form of liquor arsenicalis) arrested the progress of the disease in a horse, and enabled the animal to continue at work.

The country (principally the low-lying and swampy valleys of the Zambesi and Chobe) in which nagana is enzootic, closely resembles in its climatic conditions that in which surra is prevalent. The progress of nagana is much slower in cattle than in horses. The wild animals which inhabit the "fly" country are immune from nagana.

Koch regarded Tsetse-fly disease as identical with surra. In any attempt to establish the unity of these two diseases, we are met with the clinical difficulty that the "fly" disease occurs as a frequent and generally fatal disease among cattle, and (according to Bruce) the occurrence of the parasite is constant in the blood of the "fly" infected animal. "The marked feature in the Indian disease is the presence of paroxysms and intermissions, during which periods the trypanosoma is present and absent respectively" (*Lingard*). We cannot, therefore, accept the absolute identity of these two diseases without further information on the subject. It is possible that the comparative immunity of Indian cattle may be due to racial idiosyncrasy similar to that of Algerian sheep to anthrax. Even then, the difference in the behaviour of the respective infusoria remains unexplained. The truth probably is that although the respective parasites of surra and nagana are closely related to each other, they are not identical.

## SOUTH AFRICAN HORSE-SICKNESS.

**Nature.**—"Horse-sickness" is an infective disease which is probably peculiar to the Equidæ, and which is characterised by intense congestion of the blood-vessels, with grave alteration of the blood, so that there is an escape of a large quantity of liquor sanguinis into various tissues.

**History and Mortality.**—This disease has been known in South Africa since the year 1780. It is enzootic in various districts, and from time to time assumes, at irregular intervals of time, an epizootic character, as for instance, during the years 1780-81, 1819, 1839, 1854-55 (Cape Colony), 1888 (Natal), 1891-92 (Cape Colony), and 1893-94 (Orange Free State). The loss of horses is very great during an epizootic of this malady. In the two years of 1854-55, 64,850 horses died of it, and in 1892, 13,979 horses and 149 mules. Edington tells us that in Rhodesia the annual loss is about 90 per cent. The lung form is fatal in at least 99 per cent. of the cases. In fact, many persons who had a wide experience of horse-sickness told me, while I was in South Africa, that they never knew a case of the lung form to recover. The other varieties are said to be less dangerous to life, although in them the rate of mortality is certainly very high.

**Etiology.**—Practical observations and the elaborate researches made by Edington, leave no doubt that this disease is caused by a microbe which has not yet been isolated. We have, however, sufficient proof to reasonably assume that this organism, whatever it may be, becomes developed into a virulent condition on grass and other herbage, under the combined influence of heat and moisture. It is probable that the microbe of horse-sickness (supposing that such an organism exists) forms toxins which exert a paralysing action on the vaso-constrictor nerves, and thus causes dilatation of the blood-vessels, with consequent increase in the amount of plasma exuded into the tissues, and have also a poisonous effect on the system. "Horse-sickness appears to be a septicaemia in the further sense that death is due to the toxic effects of substances manufactured by the bacteria multiplying in the blood. No doubt in those cases in which there is very extensive pulmonary œdema, interference with the aëration of the blood will act as a

contributory cause of death, or it may be the final cause; but in a considerable number of the experiments recorded in this article, the combined structural lesions, such as the œdema of the lungs and the exudation into the pleural and pericardial sacs, were not sufficient to account for the fatal issue. It ought also be noticed that the microbe of horse-sickness does not appear to attach itself to the red corpuscles of the blood; at least, that seems to be indicated by the fact that these corpuscles appear quite normal in preparations made from the blood, and by the normal tint of the serum furnished by horse-sickness blood" (*McFadyean*). This eminent bacteriologist considers that it is highly probable that the agent of infection in horse-sickness is too small to be made visible to the human eye, even when aided by the highest powers of the best modern microscope. He supports this opinion by the fact that "horse-sickness blood is not deprived of its infective property by filtration through a Berkefeld or a Chamberland filter" (*Journal of Comp. Path.*, June, 1901), either of which might arrest some of the contained bacteria.

**Pathogenesis.**—There is practically no doubt that the virus of this disease is transmitted to the horse, as a rule, by means of *forage*, particularly when such forage is laden with *dew*. The contagium is not only fixed, but according to all South African experience, the disease is never, under natural circumstances, communicated from one horse to another. The influence of dew or damp, apparently on the development of the micro-organism, is shown by the frequent occurrence, during an outbreak, of cases of this disease among horses which are allowed to graze while dew is on the herbage, and by the almost absolute protection afforded by restricting the animals to the consumption of dry fodder. It is instructive to note that cases of horse sickness are all but unknown in South African racing-stables, the horses of which seldom, if ever, get any green grass when in training. They generally go out, however, in the early morning, while the dew is on the grass. As such protection is obtained without paying any attention to the nature of the drinking water, it would not be reasonable to suppose that water acts as a carrier of the contagium. "I have taken the water from pools in horse sickness areas during a season of epidemic and have examined it microscopically, but without success. Under the supposition that the spores under a protean guise might be present among the *débris* and multitudes of



animalculæ with which such waters abound, I inoculated horses with as much as 10 cc., but without any result accruing" (*Edington*). Arguing against the malarial character of horse sickness, Wiltshire points out "that there have been instances of great mortality from it in one district, while an adjoining one, separated only by a narrow river, has been almost entirely exempt."

The experience of South African farmers is greatly in favour of keeping horses, during an outbreak of horse sickness, in a stable, or even in a *kraal*, if it be intended to turn them out to graze, until the sun has dissipated the dew off the grass, say until eight o'clock in the morning. Many South African farmers have told me that keeping horses in a *kraal* is quite as good for preventing the sickness as stabling them. I may explain that a *kraal* is simply a walled-in enclosure, which, from being constantly trodden down by cattle and horses, has little or no grass on it.

The respective influences of *moisture* and *altitude* are well marked. The effect of dew on the development of the microbe has already been noted. Dry, high pastures at an altitude, say, of 6,000 feet above the level of the sea, are, as a rule, free from the disease, unless they have been contaminated by the introduction of affected stock; the original home of this organism appearing to be moist and low-lying lands. Many of the South African highlands which were formerly exempt, have become infected, apparently by the introduction of diseased horses. We are at present unable to say how long the microbes of horse sickness would continue to exist in a dry and cold locality, in which, we may take for granted, they would be more or less exotics. As a factor in arresting the development of the microbes, altitude above the sea seems to owe its effect to increased cold and dryness.

In almost all cases, *frost*, similar to its action on yellow fever in man, stops the progress of an outbreak of horse sickness.

The *season of the year* has a considerable influence on the spread of this malady. In Cape Colony, Orange Free State, and Transvaal the disease rarely occurs before February, although in 1892 it broke out in the eastern province of Cape Colony early in January. It is usually at its worst in April, and as a rule disappears with the first frost in May. "In Natal it generally begins on the sea-coast a little before Christmas; at Maritzburg, about February; and up country, somewhat later. March and April are the worst months. Occasional

cases are met with even in the winter on the coast. One undoubted instance occurred near Ladysmith during the Boer war in 1881, in July or August, when the ground was covered with snow. As summer is the rainy season in Natal, a hot, moist climate is supposed there to be most favourable for an outbreak of the disease, which, in this respect, does not follow any definite rule. In fact, some wet seasons have been very healthy, and some dry ones particularly deadly" (*Wiltshire*).

It is possible that *debilitating influences*, such as exposure, want of proper food, and fatigue, may predispose a horse to contract horse sickness by lessening the ability of the vital forces of the animal to resist an attack. I have been informed by proprietors and managers of various South African mail coach lines that their horses, which are well-fed, though indifferently stabled, enjoy a marked immunity from this disease, even when animals at grass in the vicinity are dying from it in great numbers. The former have to do their work at all hours, late and early. The latter, as a rule, are more or less starved. I am inclined to think that the comparative immunity in this case is derived from the restriction in diet to dry fodder, and not to the quality or amount of the food.

*Inoculation* or *drenching* with the blood of an infected horse appears to invariably convey the disease; but inoculation with the blood serum which is discharged from the nostrils does not always give positive results.

**Anatomy.**—The lungs are invaded by a yellow-coloured transudation of plasma, the presence of which can be seen on the outer surface of the lungs, upon which it appears in yellow patches. There is generally a large amount of this fluid in the pericardium and also in other parts; and the bronchial tubes are more or less filled up with it. Although the blood is darker than usual, it does not in any way possess the tarry appearance which is characteristic of anthrax. It coagulates with extreme quickness.

"On making an incision into the subcutaneous tissue of the neck, along the line of the windpipe, it is common to find lying along, and in many cases surrounding the windpipe and the larger vessels, a quantity of clear yellow jelly, which consists of blood serum that has been pressed out of the vessels in this situation, and has subsequently undergone coagulation. If the windpipe is opened, it will be found to contain yellow fluid in all

stages of conversion into the white froth that one may notice after death around the nose and mouth.

"On opening into the large blood-vessels in this area, the blood will be found to contain clots, while the fluid between may be slightly watery and is invariably of a very black colour.

"On removing the front of the chest, the pleuræ may be seen to be occupied by a large amount of blood-coloured fluid.

"On the surface of the lungs it is common to find a layer, more or less thick and diffused, of a yellow, gelatinous material consisting of coagulated blood serum. The lungs are, as a rule, pale in colour, unless the animal has been severely ridden previous to its death. On the surface will be noticed a large number of dark-coloured lines which seem to pass over it and appear like small rivulets. If slices of the lungs be made with a knife and held up to the light, it will be noticed that the lines in question are transparent and of a yellow colour. They consist in fact of the yellow serum, exuded in this situation, that has coagulated, and they occupy the lines of division between the lobules of the lungs and which in health ought to be scarcely apparent. . . . In the *dikkop* form, the chief sign is the swelling of the head, neck, or tongue. In this form the lungs may be almost or absolutely unaffected to the naked eye.

"The spleen, as a rule, is enlarged and deeply congested, and over the whole surface may be seen small hæmorrhagic points. On section, the capsule immediately retracts, and the substance is seen to be very dark in colour, owing to venous engorgement" (*Edington*).

**Varieties.**—The two ordinary forms of horse sickness are : The lung form (Dutch, *paard ziekte*), which is the most common kind ; and "thick head" (Dutch, *dikkop*). The manner in which the œdema is localised constitutes the only difference between these two forms, which more or less run into each other. "Blue tongue" is a third form which may be regarded as a variety of *dikkop*.

**Period of Incubation.**—The period of incubation is about 8 days, with probably a variation of about one day, one way or the other.

**Symptoms.**—*Edington* states that the first symptom of the lung form of horse-sickness is a shivering fit with a rise of temperature up to 39.5° C. in the evening ; that the temperature next

morning is lower, though not quite normal, and rises still more in the evening; and that it thus steadily, though slowly, increases until within a few hours of death, when it may fall below the normal standard. Before this fall, the temperature reaches to a considerable height. Towards the end there is great congestion of the blood-vessels; the mucous membrane of the eyes and nose assumes a dark red colour, and a copious inter-lobular effusion takes place in the lungs. The breathing becomes extremely hurried, often about 75 in the minute, with heaving of the flanks. Depression is well marked; and there is an escape into the bronchial tubes of serum, which becomes mixed with mucus. The presence of this serum, even before any discharge takes place from the nostrils, can be readily detected by the bubbling sound that may be heard by applying the ear to the front of the chest. As this fluid accumulates more and more in the bronchial tubes, it is discharged from the nostrils, usually in large quantities, and in a somewhat frothy condition, which it soon loses if it be allowed to collect on the ground, in which case it will be seen to be of a yellow colour. The fluid may trickle from the nostrils, or may be discharged in streams. Although I have never measured in a fatal case the quantity of this poured-out fluid, I would conjecture that it would be about 3 or 4 pints. "I have caught quantities of this material in glass vessels, when it appears as a straw-coloured fluid, but it is spontaneously coagulable in the presence of minute traces of blood. It is coagulated by heat, and has been found to consist almost entirely of blood plasma" (*Edington*). "One very characteristic symptom is the distension or bulging of the pits above the eyes" (*Hutcheon*). The froth that is discharged from the nostrils is produced by the plasma which has transuded into the air-cells of the lungs and is somewhat similar in composition to the white of an egg, becoming churned up into foam by the breathed-in air. To use Edington's happy expression, the animal is practically drowned in its own blood serum.

This disease runs a fatal course, usually in about 4 days, taking the period of incubation at about 8 days. During the first two days of the attack there is little to attract casual attention. As a rule, the patient dies very suddenly, frequently within an hour or two of the time he was first seen to be ill. The general idea in South Africa is that the disease runs its course in a couple of hours, or even less; the cause of this mistaken opinion being evidently want of ability to recognise the symp-

toms until they are of a pronounced character. As a rule, death from suffocation comes on suddenly.

In *dikkop*, the chief manifestation is swelling of the head and neck in a manner closely resembling that seen in the respiratory form of anthrax. In *dikkop* there is always more or less congestion of the pulmonary vessels; and in *paard ziekte*, more or less transudation of liquor sanguinis into the subcutaneous tissues of the head and neck. Consequently these forms are never absolutely pure.

"In the *blue tongue* variety, symptoms of pneumonia are not very prominent. The muzzle, lips, and tongue become swollen. The tongue also assumes a dark blue appearance. It turns cold, and often becomes too large to be retained in the mouth. Thick, dirty saliva falls from the mouth" (*Wiltshire*).

**Manner of Death.**—According to Edington's researches, death is not due to a microbic toxin; for "the injection into a healthy horse of a considerable amount of filtered blood serum, taken from a horse dying of horse-sickness, has not been attended, in my hands, with any subsequent symptom at all referable to or suggestive of horse-sickness." On the other hand, with inoculations of unfiltered horse-sickness blood, Edington was almost invariably successful in producing the disease.

**Differential Diagnosis.**—The only diseases horse-sickness could be mistaken for are anthrax (p. 348), congestion of the lungs, and pneumonia.

**Prophylaxis.**—During a sickly period, if the horses be at grass, they should, if possible, be sent away to some high and unaffected grazing ground above the zone of the disease, as is generally done by owners of large droves of horses who have facilities for doing so, like those at Grahamstown and Harrismith. Stabled horses should be fed on dry food, such as "forage" (as unthrashed oats are termed in South Africa) and "mealies" (Indian corn) or oats, and should on no account be allowed any grass. As a compromise between convenience and security, horses in the open should be kept in a kraal at night, and should not be allowed to graze until the sun has removed all the dew from the grass. In addition to the previous remarks made in support of this advice, I may mention that I have heard, from reliable authorities, of several cases of stabled horses, during an outbreak, contracting the disease, apparently from having been

fed on grass which was cut and freshly brought to them, while those which were in the same stable, but which were kept on dry food, remained healthy. These practical observations, strengthened by Edington's investigations, prove almost to demonstration that the contagium becomes developed into a virulent condition on grass (or other herbage) under the influence of moisture and especially of dew. Hence the best possible preventive means is the limitation in diet to dry food during a sickly season. So long as horses are prevented from grazing, no danger would seem to be incurred by keeping them out in the open while dew is on the ground.

**Treatment** has proved up to the present to be of no avail.

**Acquired Immunity.**—By methods which he has not yet made public, Edington has succeeded in rendering horses immune from horse-sickness by *inoculation*, which was accompanied by a mortality of about 1 in 3. There are no details available as to the length of this acquired immunity.

It is a common belief in South Africa that horses which have recovered from an attack of horse-sickness possess a life-long immunity from it, and that consequently their value becomes much enhanced in districts in which the malady is enzootic. During a visit to South Africa, I was able to obtain but little exact information on the subject of "*salted*" horses, which, I had imagined, were an ordinary article of commerce in that country. In fact, though I made many inquiries, I failed to find a single animal of the kind. The few persons I met who had, so they said, seen "*salted*" horses, informed me that these animals present a dejected and debilitated appearance; that the skin about their head and neck is unusually loose and wrinkled; and that they are liable to relapses of the disease, though in a milder form. Mr. Wiltshire (late Colonial veterinary surgeon of Natal) tells me that "*salted*" horses invariably die from horse sickness, if they be allowed to live long enough. From a photograph which I have before me of a "*salted*" horse, I see that the hair of his mane and forelock stick out in a particularly rough and disordered manner, so that it would have been impossible to have made the mane lie on one side, or the forelock to fall straight down. I have heard that all "*salted*" horses have this peculiarity of the hair of the mane, forelock, and tail. I am inclined to think that these so-called "*salted*" horses are those which are suffering from the disease communicated by the Tsetse-fly.

## DISEASES CONVEYED BY TICKS.

Australian Tick Fever—Ixodic Anæmia in Jamaica—Louping ill.

As far as we can judge with the knowledge at present in our possession, infective diseases conveyed by ticks (*ixodes*) to animals assume two entirely distinct forms : one, that of anæmia, as in Texas fever (p. 415), Australian tick fever, and ixodic anæmia in Jamaica (Williams); the other, that of nervous disturbance, as in louping-ill. The three maladies which come under the first heading appear to be closely-allied varieties of the same disease. The respective contagia of all these diseases seem to be ectogenous, by the fact that they cannot, as far as we know, be transmitted from one animal to another. Consequently, the cadavers are not sources of danger to man or beast.

The bovine tick-carried diseases of Texas, Australia, and Jamaica not only present points of analogy with the malarial fevers of man (p. 417), but also resemble surra (p. 455) and Tsetse-fly disease (p. 464), as far as their course, symptoms, and parasite are concerned.

## AUSTRALIAN TICK FEVER.

“Tick fever” is a bovine disease which has become widely disseminated throughout South Australia and Queensland, in both of which colonies it has caused great losses. According to the researches of Mr. C. J. Pound (Government Bacteriologist), Dr. J. Sidney Hunt, and Mr. William Collins (see *Report on Tick Fever*, Brisbane, 1896), tick fever is identical with Texas fever. Formerly it was known in Queensland as “Redwater Disease,” which was an inappropriate designation ; because hæmaturia has no necessary connection with this malady and is not a constant symptom of it. Smith and Kilborne found hæmaturia in 33 out of 46 cases of Texas fever, and state that “one sign regarded as peculiar and pathognomonic in this disease is the discharge of urine having the colour of blood.” The hæmaturia present in cases of tick fever, and also in those of Texas fever, appears to be due to destruction of red corpuscles of the blood by the specific hæmatozoa ; the result being that the hæmoglobin set free from the disintegrated corpuscles passes into and, if in sufficient quantity, discolours the urine. Hence, when the destruction of the red corpuscles has been comparatively slight, no perceptible inter-

ference with the normal colour of the urine may occur. We are unable to say that the course of the disease and the resulting anæmia is due to such destruction, or to some other cause, such as, for instance, a toxin. Not only were ticks which were taken off infected cattle proved to have been capable of transmitting the disease, but young ticks which were incubated from them were also shown to possess the power of communicating tick fever to cattle upon which they were placed.

The hæmatozoa of tick fever are similar to those of Texas fever, and are found on a greater or less number of the red corpuscles, and also in a free condition in the liquor sanguinis. The free or extra-corpuscular bodies are comparatively rare in the circulating blood, but are abundantly present in the capillaries of the kidneys and spleen. They are from  $0.3\ \mu$  to  $2\ \mu$  in length. Although most of them are round, they vary greatly in shape, as they keep constantly changing their form. They are highly refractive, and generally colourless, homogeneous and motile. Some have a yellow or reddish brown tinge, but without any granular pigment. A few are non-motile, "but the majority are in very active movement, and may sometimes be seen to work their way across the field of the microscope, apparently urged along by a flagellum. The most general and characteristic movement, however, is neither amœboid nor locomotive, but consists of a peculiar rolling on their own axis, which gives them a twinkling appearance, something like that of a small bright coin as it sinks in deep water" (*Report on Tick Fever*).

The intra-corpuscular forms are pale, rounded bodies which occupy about a fifth of the invaded red corpuscle.

Attempts to cultivate these organisms have been unsuccessful.

Sheep and horses have been proved to be immune.

In districts in which this disease is enzootic, cattle often acquire immunity from having been constantly infected by ticks.

"The points of analogy between tick fever and the malarial fevers of man are so many and obvious as at once to arrest attention. Both are due to amœboid blood parasites which occur as intra and extra-corpuscular forms; in both there is a long continuance of the micro-organism in the blood; both present themselves in acute or malignant and in chronic or mild forms; both are characterised by blood destruction and enlargement of the spleen; both are liable to recur; and lastly, both are associated with insects" (*Report on Tick Fever*).



For a summary of the *Report on Tick Fever*, see *Journal of Comparative Pathology and Therapeutics*, March, 1897. For information on protective inoculation experiments with the blood of cattle which have recovered from this disease, see *Queensland Agricultural Journal*, August, 1897.

#### IXODIC ANÆMIA IN JAMAICA.

The late Professor Williams (*Principles and Practice of Veterinary Medicine*) made a careful study of this disease, and identified it with Texas fever, with the exception of a few trifling differences, due apparently to local influences.

The chief symptoms observed by Williams were great debility, depression, anæmia, and emaciation. He found, contrary to what is the case in Texas fever, absence of enlargement of the spleen, and absence of hæmaturia, with the exception of one doubtful case. It is satisfactory to learn from recent reports that the prophylactic measures recommended by Williams have been productive of great economic benefit to the island. They were identical in principle to those which are advised in the prevention of louping-ill (see below), and consisted in depriving the ticks of cover as far as possible; in liming and improving the land; and in dressing and dipping the cattle. For details on this subject, readers are referred to Williams's work on Veterinary Medicine, and to his *Report* which appeared in the *Veterinary Journal*, December, 1896.

#### LOUPING-ILL (*trembling*).

**Definition.**—Louping-ill is an infective disease which is almost entirely confined to sheep, and which is characterised by symptoms of nervous derangement. Dr. Hamilton of Hawick considers that it "is primarily a congestion of the brain and spinal cord."

The term "louping-ill" (old Scotch, *loup* = leap) is derived from the fact that sufferers from this disease will often, if suddenly startled, leap into the air, and will then fall down in a fit, during which they will exhibit more or less severe symptoms of this malady.

**Occurrence.**—Although louping-ill is essentially an ovine disease, it has been observed in horses by Williams; and "swine fed with the carcasses or blood of sheep which have succumbed to louping-ill die with every characteristic of the disease in a short time. If the carcass has been boiled, this does not occur.

Swine will also take the malady if allowed access to the grass of affected fields" (*Meek and Greig Smith*). It is a stationary disease, the limits of which in various districts are often sharply defined. In England "it is confined to the North Tyne district of Northumberland, and extends into Kirkcudbrightshire and certain valleys of Dumfriesshire. It is rare in Berwickshire, common in the north and west of Roxburghshire, and the similar hilly districts of Selkirkshire and Peeblesshire. It occurs in Ayrshire, to a slight extent in Lanarkshire, and is found in the western parts and islands of Argyleshire and Inverness-shire" (*Report of the Louping-ill Committee of the Teviotdale Farmers' Club, 1880*). It is more common among lambs than among full-grown sheep. It usually begins in April and ends early in June. "In Skye there are two annual outbreaks; one in early summer, and another in autumn, commencing at the end of August and terminating in a month or six weeks" (*Williams*). "We have been told of instances of sheep taking the disease through being simply driven over affected fields, and this statement is in accordance with the *Report of the Teviotdale Farmers' Club Committee*" (*Meek and Greig Smith*). Sheep bred on infected pastures acquire a certain amount of immunity.

**Symptoms.**—Reflex irritability is greatly increased; the temperature is raised; rate of pulse and respiration accelerated; and the patient exhibits various forms of nervous disturbance, such as convulsions, spasms, paralysis, trembling, squinting, and wry neck. The spasms in some of the muscles are tonic, in others, clonic. The more or less characteristic trembling is due to clonic spasms.

"The disease can be recognised by the more or less complete paralysis of the body and limbs. Symptoms may succeed one another very rapidly, or may spread over some length of time. The animal at first loses control over the muscles, which are seen to twitch convulsively. It may fall down and struggle on the ground, sometimes jumping up again, often to some height. Between the fits it is often seen to stand trembling. These symptoms are frequently accompanied by frothing at the mouth. Some such appearances are the usual outset to the disease, and are followed by a paralysis which usually affects the hind limbs, but may also include more or less of the body and head and neck. The fore limbs are often similarly paralysed. The affected limbs become cold to the touch. The paralysis neces-

sarily brings the animal to the ground, though it may be able to crawl about by the aid of the unaffected legs. When the head and neck are affected, the former is usually drawn to one side, and the eyes often become oblique. Excitement is greatly increased when the animal is disturbed. The symptoms then, in a few words, are more or less complete paralysis, preceded as a rule by fits and trembling.

"The small number which recover present a 'wry neck, stiff joint, high back, or other deformity' (*Hawick Express*). During recovery, swellings occur at the joints; these may be pierced with good results, giving a large discharge of pus. According to Fair (*Veterinarian*, vol. viii.), 'these abscesses usually appear in the neighbourhood of the joints, but sometimes above the arms, the brisket, or any neighbouring part of the body' " (*Meek and Greig Smith*).

**Etiology and Pathogenesis.**—Williams, who has done much good work in the investigation of this disease, has formed the well-reasoned-out opinion that ticks are the vehicles of the virus. The conclusions at which he has arrived on this subject are as follows: "(1) That where louping-ill existed, there the tick was sure to be. (2) That where the parasites were absent there was no louping-ill; and it is a remarkable fact that upon ground having a westerly or southern aspect ticks and louping-ill prevail, whilst on adjoining grounds with easterly or northerly aspects, upon which the grasses are short, thus providing no cover for the ticks, the disease is rare. (3) That the appearance of the parasites on the sheep in April and early summer was concomitant with the annual outbreak of the disease. (4) That in Skye, where it is said the disease appears both in spring and autumn, the tick being also there in autumn as well as in spring." Although the connection of ticks with louping-ill appears to be absolute, the fact of ticks being found all over the world on land, free from louping-ill, is a strong reason for thinking that the ticks are only a vehicle for the virus. Williams further notes the interesting circumstance that the destruction of the natural cover of the ticks by ploughing up and liming the old grasses is followed by the eradication of louping-ill.

The following experiment, conducted by Meek and Greig Smith (*Veterinarian*, December, 1897), supports to a certain extent the conclusions of Williams as to the pathogenesis of the disease. These two observers turned out on louping-ill infected ground 20 sheep, 6 being muzzled so as to prevent them

from feeding on the grass of that land, 7 being dressed with a composition (sweet oil, 2 quarts; castor oil, 1 quart; train oil, 1 quart; pitch oil,  $\frac{3}{4}$  pint; and cade oil,  $\frac{1}{4}$  pint) calculated to keep off ticks, and 7 being allowed to graze without being either muzzled or dressed. Of the 6 muzzled ones—which were fed night and morning on hay and turnips obtained from farms which never had the disease—2 died of louping-ill; there was one casualty from this disease among the 7 which were neither muzzled nor dressed; but all the 7 dressed sheep remained healthy.

Meek and Greig Smith have found that the tick inoculates two pathogenic germs: one a pus organism; the other, a bacterium which produces in rabbits tetanic spasms and *post mortem* appearances, similar to those of *louping-ill*.

**Differential Diagnosis.**—Anthrax and braxy are often mistaken for louping-ill. I would suggest the same possibility with regard to tetanus, which may be distinguished by the freedom from cerebral disturbance and the tonic character of the spasms of particular groups of muscles. The alterations due to cerebral and spinal congestion found, *post mortem*, in louping-ill cases, are absent in those of tetanus, in which there are little or no pathogenic changes. In both anthrax and braxy there is rapid decomposition of the cadaver, contrary to what occurs in louping-ill.

**Anatomy.**—The pathological changes are chiefly limited to the brain and spinal cord, and generally consist in congestion and inflammation of the meninges, and increase in the amount of the cerebro-spinal fluid. The pleuræ and pericardium are often affected with resulting serous effusion. Williams states that the flesh is usually pale, and that as a rule no really characteristic lesions are to be found. The cadaver resists decomposition for a comparatively long time.

**Prophylaxis.**—Meek and Greig Smith, agreeably to the observations of Williams, point out that as the larvæ of the ticks die if they do not find a suitable host in early summer, the farmer would do well to burn down the old withered grass on infected lands during the autumn, and to keep sheep off such pastures as long as the louping-ill season continues. The advisability of breaking up and liming the land, as recommended by Williams, or dressing it with salt, is self-suggestive after we

have grasped the fact that the tick requires an animal host in at least one period of its development.

The length of time necessary for an application to remain effective, militates against the benefit to be obtained from dipping and dressing as means for destroying the ticks on the sheep, or for preventing these parasites from lodging on the skin of these ruminants.

**Prognosis and Treatment.**—The absence of statistics on the subject renders it hazardous to say more with respect to the rate of mortality, than that recoveries from louping-ill are not unfrequent. Meek puts the average percentage of deaths at from 10 to 20 of the whole flock. No course of successful treatment has as yet been devised.

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#### ULCERATIVE LYMPHANGITIS.

**Etiology.**—This disease is caused by a specific bacillus, which is an aerobe, and was discovered by Nocard. These microbes, which are found in large numbers in the pus issuing from a recently-opened abscess of this disease, are short thick rods which have rounded ends and are generally placed parallel to each other, although sometimes they are arranged in lines. They differ from the rods (bacilli) of glanders by the fact that they stain by Gram's method.

**Diagnosis.**—A bacteriological examination of the respective microbes easily distinguishes this disease from glanders and epizootic lymphangitis. In ulcerative lymphangitis, abscesses do not form in the ganglions, and although the attacked ganglions become infiltrated, they do not become hard. In the other two diseases, abscesses form in ganglions. Mallein gives no typical reaction in cases of this disease.

**Symptoms.**—The disease usually breaks out in one of the legs; the hind being more commonly affected than the fore. The lymphatic vessels of the suffering limb swell and form abscesses, which on bursting give rise to deep, unhealthy-looking ulcers on the inside and sometimes on the outside of the leg, which may become swollen and the animal may not be able to put weight on it, or even allow it to rest on the ground. In

such cases, pressure on the limb will cause the patient great pain. Abscesses and consequently ulcers may form on the lower part of the body. Sometimes the abscesses and the corded condition of the lymphatics disappear and the ulcers heal up in the summer, only to come on again during the next cold weather. Recovery may take place spontaneously, or the attack may continue for several years. Nocard lays great stress on the fact that in all cases which he has seen, the lymphatic glands of the groin of an affected limb never became hard or had abscesses, although occasionally they might become somewhat enlarged.

*Post-mortem* examination often shows that suppuration has extended to the kidneys, but never to the liver, spleen or lungs.

Nocard considers that ulcerative lymphangitis is not easily transmitted from one animal to another, and that consequently it is not a particularly dangerous disease.

**Treatment.**—Scrape the wounds, and apply antiseptics.

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#### NOTES ON EPIZOOTIC LYMPHANGITIS (p. 288).

Formerly this disease was common in France, but it is no longer met with in that country. Dr. Lingard recognised it in 1899 among horses at the Indian Remount Depôts of Karnal and Hapur, where it had been mistaken for farcy, with the result that many remounts were needlessly destroyed, although tests with mallein gave negative results. In this case, it had been imported by mules from Italy.

The microbe of this disease (*cryptococcus farciminosus* of Rivolta) is about one six-thousandth of an inch in diameter. When pus from a freshly-opened abscess is microscopically examined with a power of 400 or 500, these organisms appear as "rounded bodies with somewhat pointed ends, or one end pointed and the other rounded, highly refractile and presenting a double contour. At first sight, one is immediately reminded of the likeness of this organism to the *coccidium oviforme* so frequently observed in the liver of the rabbit, or of a yeast cell, *sacchromyces*" (Lingard). This microbe readily stains by Gram's method. Cases of this disease do not respond to the mallein test.

With reference to the occurrence of this disease in India, Dr. Lingard states in his *Annual Report* for 1900-1901, "that

experiments conducted at Muktesar have shown that a cure can be effected by removing animals to a high level, 7,500 feet elevation, a result which agrees with the fact that the disease in Europe disappears above a given line of latitude. As the development of the disease occupies several months, this measure can easily and usefully be adopted in the case of valuable remount animals."

"The horse, ass, mule and the bovidæ are susceptible to this disease; but the dog, cat, calf and pig are immune" (*Cagny and Gobert*).

Epizootic lymphangitis is easily transmitted from one horse to another.

## CHAPTER V.

## NOTES ON BACTERIOLOGY.

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THESE Notes are intended to assist those readers of this book who are not conversant with bacteriological methods, to understand the bacteriological portion of the text. They are in no sense intended to be a detailed statement on the modern methods of bacteriology.\*

**Classification.**—Pathogenic microbes belong either to the vegetable or to the animal kingdom. The only pathogenic organisms belonging to the latter are certain protozoa, such as the plasmodium malariae of Laveran (now known as the *Hæmamoeba*), the surra infusorian, and psorospermia or coccidia, which are "unicellular parasites of fixed form of body, surrounded by a capsule, forming within their body a number of spores, each surrounded by a cuticle, the spores becoming free after the bursting of the capsule, and giving rise to a new parasite." All these protozoa closely approach the vegetable kingdom.

Pathogenic vegetable microbes, which form the vast majority

\* There are many excellent manuals on bacteriology; one written especially to meet the requirements of veterinary surgeons being *Bacteriology and Infective Diseases*, by Professor Crookshank. Among the smaller works, *A Manual of Bacteriology*, by Muir and Ritchie; *A Manual of Bacteriology*, by Professor R. T. Hewlett; *Essentials of Practical Bacteriology*, by H. J. Curtis; *Bacteriological Technique*, by J. W. H. Eyre, are all excellent and reliable. The veterinarian concerned with the examination of milk may find *Bacteriology of Milk*, by Swithinbank and Newman, a useful book.



of disease-producing germs, belong to the *protophyta* (plants that have no sexual reproduction), and are divided into : (1) *bacteria* ; (2) *yeasts* (*saccharomyces*) ; and (3) *moulds*. Although yeasts and moulds play a large part in the functions of animal life, few of them are actual disease producers. As instances to the contrary, certain pathogenic yeasts, *oidium albicans* and the fungus of favus (a mould) may be cited. *Moulds* consist of branched filaments (*hyphæ*), which form a felt-work (*mycelium*) by becoming interlaced. *Conidia* (spores of moulds) become developed at the ends of the threads (*hyphæ*).

Bacteria, yeasts and moulds are distinguished from each other by their respective *methods of multiplication*. Thus, bacteria multiply by division ; yeasts by budding ; and moulds by branching.

The Protophyta belong to the Thallophyta (plants that are not differentiated into stem, root, leaf, etc.), which are a sub-order of Cryptogams, the vegetable kingdom being divided into Cryptogams (flower-less plants) and Phanerogams (flowering plants).

For microscopic purposes, bacteria are usually classified as follows :—

1. Rods (*Bacilli*) whose length is greater than their breadth and whose sides are parallel.
2. Round cells (*Micrococci*).
3. Spiral forms (*Spirilla*, *Vibrones*, or *spirochæta*), which are curved rods that, on uniting, form wavy S-shaped, chain-like, or spiral threads (filaments).

The term *bacterium* (Gr. *βακτήριον*, a small rod) is now used, especially in the plural, as a generic name to cover all the micro-organisms, whether rods or round cells, and the term *bacillus* is confined to those which are strictly rods.

Bacteria may be divided according to a variety of characters or conditions. If they be classified according to their condition of growth in respect of oxygen, the classification would be as follows :—

1. *Ærobes*, which, like *Bacillus subtilis*, require air (oxygen) for the maintenance of their life.
2. *Anærobes*, which, like the bacilli of tetanus, malignant œdema and symptomatic anthrax, cannot live in the presence of oxygen.

*Facultative anærobes* grow moderately well under a free supply of air, or when oxygen is entirely excluded. To this group belong most pathogenic organisms. *Obligatory ærobes*

cannot, or can only to a very slight extent, grow without oxygen. *Obligatory* anærobes are unable to grow, or only feebly, unless oxygen is excluded.

If, on the other hand, bacteria be divided according to their method of nutrition, the classification would be as follows:—

1. *Parasites*, which feed on, or at the expense of, another living organism (animal or vegetable), and which may be divided into (a) *obligate parasites*, and (b) *facultative parasites*. The former, like the bacillus of leprosy, can grow only in their animal host. The latter, like the bacillus of anthrax, are able to vegetate both in the animal body and apart from it. Most pathogenic bacteria are more or less true parasites.

2. *Saprophytes*, which obtain their nutriment from dead organic matter, play an important part in the economy of nature in breaking down complex substances into simpler compounds, which may again be taken up and assimilated. Thus carbon and nitrogen are supplied to the higher vegetation which is necessary for life in the animal kingdom. Hence the majority of bacteria, being saprophytes, must be looked upon as essential to life. In a limited sense all the parasitic bacteria which can be subcultured outside the body are saprophytic.

Micro-organisms may also be divided into pathogenic (or disease producers) and non-pathogenic; or according as to whether they liquefy gelatine or not; or by their products; or by their behaviour in artificial media; or according to the method by which they are stained; etc.

Further, it has been observed that bacteria may be classified not only by the form of the individual elements but also by their respective mode of arrangement to each other. These different arrangements and groupings are respectively peculiar to different species. For example, a *diplobacillus* is formed by the transverse division of a bacillus into two parts which continue to remain together; *leptothrix* consists of long rods or threads which have no branches; and *cladotrix*, of long rods or threads which have branches. A *micrococcus* is a single small round cell; the *diplococcus*, two divisions of a single coccus with or without a capsule (as in Diplococci of Pneumonia and Gonorrhœa); a *streptococcus* is a chain of cocci each more or less a part of its neighbour, in longer or shorter chains, and sometimes in rosaries, as in Streptococcus of Pus and Erysipelas. The *Staphylococcus* (Gr. *σταφύλις*, a bunch of grapes) is a mass of round cells as occurs in the various Staphylococci of Suppuration. *Sarcinæ*

are packets of cocci in one plane, and *tetrads* are small square masses of cocci produced by division into four parts in two planes at right angles to each other. Again, we have different elements, or different arrangements of elements, having further characteristics—*e.g.*, many of the bacilli have terminal or lateral *flagella*, which are long or short threads of protoplasm, not staining readily, and by their vibration causing progressive movement of the bacillus (*e.g.*, *Bacillus Typhosus*). There is another marked difference which must always be borne in mind, namely, that due to polymorphism, which, amongst other things, gives rise, under abnormal conditions, to *involution forms*. These are degenerate forms and assume various shapes and sizes, diverging in a high degree from the normal. All these characters and modifications may and do serve as bases for classification. A convenient general classification is into the Lower Bacteria (cocci, bacilli, and spirilla), and Higher Bacteria (*Leptothrix*, *Cladothrix* and *Streptothrix*—one of the most important of which group is the *Streptothrix Actinomyces*).

**Biology.**—Bacteria are composed of a body of more or less homogeneous protoplasm, though many substances may be found therein. Sulphur, starch, and, in some organisms, pigment have been observed. Amongst the last-mentioned may be cited *Spirillum rubrum* (which is exceptional in not requiring free oxygen for its pigmentation), *M. prodigiosus*, *B. pyocyaneus* (except when cultured at 44° C.), and *B. violaceus*.

In addition, we may frequently detect in the body of a bacillus small granules which, owing to their staining propensities, are called *meta-chromatic granules*. They should not be confounded with *polar bodies*, which are occasionally found at the extremities of bacilli or spirilla, and which, in their turn, should not be mistaken for *spores*. Nencki has pointed out that 85 per cent. of the substance of the body of a bacillus is composed of albuminous matter which he termed *mycoprotein*. Around this there is frequently a capsule of cellulose.

Bacteria are reproduced by fission or spore formation. The micrococci multiply by simple division, and, generally speaking, bacilli do the same, though they never divide longitudinally. The method of fission is simply that a slight indentation occurs which becomes a more and more marked constriction up to the point of separation. "Spore Formation" may be either inside the capsule of the bacillus—*endospore*—or outside as part of the chain of cells—*arthrospore*. Endospore formation is well seen in the

bacillus of Anthrax. The protoplasm becomes granular and the small specks enlarge into encapsuled, oval, sharply-defined bodies growing at the expense of the protoplasm of the bacillus. Eventually, either by exhausting the bacillus or bursting its capsule, the spore escapes. It fulfils its function like a seed, not producing other spores but germinating into a small bacillus. The capsule or the concentrated protoplasm of the spore, or both, afford it a very high degree of protection from inimical influences, such as desiccation, heat, or chemicals; especially is this the case with the spores of Anthrax, which can retain life and virulence in a dried condition for weeks and even months. Spores may occupy different positions in the bacillus in different species—for example, in the bacillus of Malignant Œdema or of Quarter Evil, the spore is generally situated nearer the end of the rod than the middle. We have also truly *terminal* spores in the bacillus of Tetanus, which then simulates a drum-stick or tin-tack. Again, though bacilli of which Anthrax is the type, have spores of a less diameter than themselves, the bacillus of Malignant Œdema produces a spore which is of greater diameter than itself and which bulges out the bacillary capsule. It is important to note that spore formation is not so much a multiplying stage as a resting one. One individual does not produce more than one spore and that spore only produces one bacterium.

Bacteria are motile or non-motile. The motility may be considered as progressive or non-progressive. The former is due to flagella either lateral, or terminal, or both, as in bacillus typhosus. The non-progressive is undulatory or rotatory, but without any advance being made. All movement is accelerated by heat, and inhibited by cold or colouring reagents.

**Products of Bacteria.**—The following are among the chief products of bacterial life :—

1. *Pigment.*—These chromogenic organisms as a general rule possess little or no pathogenic action. Light and oxygen are generally necessary for the production of pigment.
2. *Ferments*, such as the peptic ferment, or that which splits up sugar, or that which coagulates casein in milk.
3. *Gases*, such as carbonic acid or methane.
4. *Phosphorescence*, which occurs mostly in sea-water organisms.

5. *Chemical Products of the nature of Toxins*: soluble substances produced by the bacteria and gaining access to the blood or lymph stream.

In the causation of disease by micro-organisms there are therefore these two main factors: *the living organisms*, multiplying and passing in many cases throughout the body; and *the chemical poisons* produced by them and which act locally and generally, and react again upon the organisms which have been their source. In some cases the organism remains more or less local, as in the diseases of Tetanus or Diphtheria, and from its local situation produces its toxins, which by means of the circulation are carried throughout the body, bringing about the clinical symptoms and signs of the specific disease they represent. In other cases, like Anthrax, the bacilli themselves pass to all parts of the body and are found even at its peripheral portions producing their poisons. It is true the organisms and poisons, either separately or together, may and do cause tissue change. But it must not necessarily be supposed that the poisons are merely secretions of the living bacteria and would occur in the same way outside the body; for in many cases they are formed indirectly by the medium of ferments, and are always influenced by, even as they influence, the living tissues of the body which is invaded. As bacteriology has advanced, emphasis has in a marked manner been placed less upon the organisms *per se*, and more upon their products. The action of bacteria, as mechanical irritants, plays a very small part in the processes of disease.

Muir and Ritchie have pointed out the two-fold effect of bacteria in the tissues:—(1) the effects of the bacteria themselves resulting in two characteristics, viz.: *tissue change and symptoms*. The former occurs locally at the seat of inoculation, and is of the nature of inflammatory reaction, acute or chronic. Sometimes the lesion has a special site, as in Typhoid Fever or Diphtheria; in other cases, like Tubercle or Anthrax, it depends upon the point of inoculation. The final result in the tissue change will of course depend upon the dose of bacteria, as regards quantity, quality and specificity, and also upon the resistance or otherwise of the tissues; it may be a comparatively transient inflammation or actual degeneration and necrosis. (2) The toxins produced by pathogenic bacteria bring about the specific characteristics of the disease, *e.g.*, in Diphtheria or Tetanus. These toxins are, as has been pointed

out, not necessarily mere secretions of the bacteria ; but are the result of the effect of ferments produced by the bacteria.

It may be well here to remark that the association of germs with disease is not of recent origin. Long ago they were found to be present in anthrax and other diseases, but their mere presence in the blood or tissues is not of course sufficient evidence of their being the actual cause of disease. It is now generally agreed amongst bacteriologists that the following conditions (Koch's postulates) must be fulfilled before any micro-organism is held to be the specific cause of any particular disease :—

1. It must always be found when the disease is present.
2. It must be isolated, and cultivated outside the animal body.
3. It must, on inoculation, produce the disease.
4. The same micro-organism must be separated from the newly infected animal.

Latterly, the plan of obtaining the toxin and producing the disease by its means, is coming to the front as an additional test of the specificity of an organism.

**Channels of Infection.**—The possible modes of infection are as follows :—

1. *Hereditary transmission*, by which organismal diseases are conveyed to the foetus from the maternal circulation, is on record, but it is obviously a channel in which there is little or no possibility of investigation.

2. *Inoculation*. Many diseases are artificially inoculable, but few are spread by this means. One of the best examples is malignant pustule (Anthrax). The commoner methods of experimental inoculation adopted in bacteriological investigation are the following : (a) *intravenous* ; (b) *intra-peritoneal* ; (c) *intra-ocular* ; and (d) *subcutaneous*, generally in the groin, and used in tuberculosis as a test of the presence of the bacilli in milk, etc. *Spraying* fine solutions of organismal culture into the trachea or nostrils and *ingestion* of food contaminated with bacteria or their toxins are methods which are adopted occasionally.

3. By *contagion*—or *entrance through an unbroken surface of skin*. This is open to question, and probably further knowledge respecting the propagation of disease will diminish the number of those spread in this manner.

4. By the *respiratory tract*: this channel of infection is supposed to be frequently the means of spreading anthrax, tubercle, diphtheria, scarlet fever, and similar diseases. Its consideration opens up the large question of micro-organic life in the air. We can only mention one or two of the outstanding facts respecting this wider question. Tyndall\* was one of the first to record the common existence of bacteria in the air and their relation to wet and dry surfaces. Dr. Russell of Glasgow† applied Tyndall's researches to practical sanitation. He pointed out that moist surfaces do not give off solid bodies "either by evaporation or under the influence of air currents"; that the locomotion of micro-organisms in the air is passive, not active; and that all organisms, even those which are microscopic, obey the law of gravitation. If these three axioms are true, it is obvious that in still air surrounded by damp surfaces we have, practically speaking, no organisms at all. And approximately this is so. Applying these facts to infectivity through the air we may understand that mucus, discharges, wet excreta, wet sputum, and all other similar vehicles of bacteria will not, so long as they are moist, part with any virus to the surrounding air. If the air is periodically or habitually infective, it is made so by desiccated liquid discharges or cutaneous exfoliation. But so long as the vehicle of micro-organisms is moist, it will not disseminate germs in the air. Manufacturers act upon this principle in their treatment of anthrax-loaded material. Before being disturbed, a bale of infected hair is immersed in water and allowed to soak, and the bale loosened in order that the water may gain access to every part. The workers are not allowed to touch the hair until the bacillary dust becomes wet, and they are required to sort out the hair while it is in that condition. Thus a dangerous process may be conducted in comparative safety. When infective dust becomes turned into mud, it will not spread disease.

Further than this, even supposing the air to be heavily laden with germs, it does not necessarily follow that they will obtain entrance to the human tissues. Professor Sims Woodhead has pointed out that adenoid tissue, if its cells are active, whatever its situation, is a means of protection, for the reason that it contains an enormous number of active cells which are capable of taking up large numbers of micro-organisms and destroying them.

\* *The Floating Matter of the Air.*

† *Jour. of Royal Institute of Public Health*, Sept. 1896, p. 407.

There is, as is well known, a ring of such lymphoid tissue surrounding the entrance to the trachea, and another round the entrance of the oesophagus in man and other mixed-feeding animals. Yet, as Woodhead has further pointed out, it is possible—especially perhaps in pigs—that this lymphoid tissue may act as a medium for the conveyance of the disease from the outer surfaces to the tissues beneath. However that may be, it is now accepted as an established fact, that the contained air within a moist perimeter is practically free from organisms and non-injurious as far as infection is concerned. But when discharges, etc., become dry, then the respiratory tract is the common channel of infection.

5. By the *alimentary canal*.—Water, milk, and foods are three of the commonest vehicles for bacteria. Typhoid, cholera, epidemic diarrhoea, tuberculosis, suppuration, actinomycosis, anthrax and many other diseases have at different times been conveyed in the food of man or animals. Probably the commonest habitant in the alimentary canal is the almost ubiquitous *B. coli communis*, which in man must be differentiated from *B. typhosus*. It need scarcely be pointed out that although pabulum for organismal life is abundant in organically polluted water and milk, it is practically non-existent in the air.

In all bacterial diseases it is desirable to detect if possible the locality and mode of entrance of the specific organism to the tissues.

**Methods of Bacteriological Examination.**—Broadly speaking there are three chief means by which knowledge is gained respecting bacteria, viz.: (a) Examination by the microscope; (b) Cultivation on artificial media, and (c) Inoculation of animals. By the first means, *morphological* facts are obtained; by the second, something is learned of the *biology* of the organism; and by the third, facts are elicited respecting its *pathogenesis*. A few notes will be added on each of these lines of investigation.

#### I. EXAMINATION BY THE MICROSCOPE.

Micro-organisms may be examined under the microscope\* in their natural unstained condition, or stained in such manner as to demonstrate their form, etc.

\* A good microscope is essential. It should have objectives of 1 inch,  $\frac{1}{2}$ , and  $\frac{1}{4}$  (oil immersion). A white light, and proper adjustment of the substage condenser and draw-tube are also necessary. A lens of  $\frac{1}{4}$  inch focal depth is the usual power required for the study of bacteria, although in some cases a lens



(a) *Unstained specimens* are readily made by placing a drop of the fluid to be examined on an ordinary slide and superimposing a cover-glass; or if it be a particle of solid substance (such as a culture growth), it may be taken and mixed with a drop of distilled water or caustic potash solution. Fresh specimens are examined in this way for determining the form and presence or absence of motility.

(b) *Stained specimens.* The microscopical examination of bacteria is greatly facilitated owing to the fact that their protoplasm readily takes up basic aniline dyes.

A very small quantity of a fluid or pure culture, or blood, sputum, discharge, etc., to be examined is placed upon a clean cover-glass or slide preparatory to microscopic examination.

By means of a platinum needle, sterilised in the flame, a minute particle of the material to be examined is spread over the cover-glass or slide in a very thin film, using if necessary a drop of distilled water. The cover-glass is then taken up with a pair of forceps and passed, with the film side upwards, through a Bunsen flame, in order, by coagulating the albumen, to make the material adhere firmly to the cover-glass. By this means the film is *fixed*. Two or three drops of the filtered staining solution (*e.g.*, gentian violet, methylene blue, fuchsin, or any aniline dye) is placed on the film, which in two or three minutes

of a focal length of  $\frac{1}{16}$ th inch, or even stronger, is desirable. Steptothrix actinomyces, which belongs to the Higher Bacteria, is better seen with a power of  $\frac{1}{8}$ th inch than with one of  $\frac{1}{16}$ th inch. The principle of the immersion lens is the filling up of the space between the lens and the cover-glass with a material whose refractive index is the same as that of the lens, so that there will be no loss of illumination by the rays of light passing through media of different powers of refraction, while proceeding from the object to the lens. The power of a microscope varies not only according to that of the lens, but also according to the power of the eye-piece. Thus the magnifying power of a 1-inch objective in a Swift's microscope varies, according to the strength of the eye-piece and to the fact that the draw-tube is closed or extended, from 25 to 140 diameters; a  $\frac{1}{8}$ th inch objective, from 175 to 690 diameters; and a  $\frac{1}{16}$ th inch objective, from 385 to 1,627 diameters. As a high-power lens gives a picture which has comparatively very little "depth" of focus, it is necessary to place the object under examination in as nearly the same plane as possible. Hence the material to be investigated should be reduced to an *extremely* thin film. The object should also be in the optical axis of the instrument, and secured in position by means of the \*spring clips. In using the oil immersion lens the body tube of the microscope must be screwed down until the lens is in contact with the oil, and nearly touching the cover-slip. The substage condenser must be screwed up flush with the stage. The best light must be obtained by adjustment of the mirror, and fine focus must be used. A skilful use of the microscope depends, of course, upon an understanding of its parts, and upon practice.

is rinsed in water, dried in the air, and mounted in Canada balsam. Such is the method of simple or single staining.

It may be necessary, however, to use the methods of staining which have the advantage of contrast colouring, staining the organisms one colour and the surrounding tissue or cells another. There are many methods for doing this; but the two simplest are those of Gram and Ziehl-Neelsen, which may be shortly described as follows:—

In employing *Gram's method* for staining tissues we place before us five glass capsules, respectively containing a solution of aniline gentian violet,\* a solution of potassium iodide in iodine, alcohol, a solution of eosin, and clove-oil. When it is required to stain a section, it is taken up with a glass needle, and placed in the aniline gentian violet and left in that capsule for ten minutes; after which it is transferred without washing into the iodine solution until it is the colour of a dry tea-leaf (blue-black), which generally takes about two minutes. After this it is placed in the alcohol until decolourised. While the section is in this solution it should be turned and returned in order to facilitate the decolourising process; but in the case of an actinomycotic section, it is important not to shake any of the "rays" out of their nests. When the colour has left the section to a sufficient extent, it is placed in the eosin solution for one and a half to two minutes, and, after being dipped for a second into the alcohol, it should be placed for about a minute in the clove-oil. Having removed it out of the clove-oil with a section-lifter, the section is placed on a glass slide, and dried by means of a piece of doubled blotting-paper. A drop of Canada balsam is dropped on the section, and finally a cover-glass is superimposed. The parts played by the fluids in the capsules are as follows: The aniline-gentian-violet stains the bacteria purple; the iodine fixes the colour in the micro-organism, creating a chemical combination and forming a blue-black pigment; the alcohol decolourises everything except the bacteria; the eosin stains the tissue a light pink, and thus produces a counter-stain; and the clove-oil clears the preparation.

Tissues containing the bacillus of anthrax, streptothrix

\* Aniline gentian violet is made by adding one part of a saturated solution of gentian violet in alcohol to ten parts of aniline oil water. The iodine or *Gram's solution* has the following composition:—Iodine one part, potassium iodide two parts, distilled water 300 parts.

Eosin may be used in strength of 1000 of water.

actinomyces, or *streptococcus pyogenes bovis*, are generally stained by Gram's method.

It will be understood that the above is the method of Gram adapted to the staining of tissues. It is, however, still more used in general practice in the staining of slide specimens. The method for this purpose is as follows :—

Place a small droplet of distilled water in the centre of a thoroughly clean slide. With the point of a fine platinum needle take up a *trace* of the culture under examination and inoculate with it the water on the slide. Spread the droplet, which now ought to have a cloudy appearance, in a thin even film by gently moving the needle in a circular direction. Dry by waving the slide to and fro in the heated air above a Bunsen burner or spirit lamp. A well spread film should have the appearance of a thin opaque cloud of even density, just visible on the surface of the slide. The staining procedure is then as follows :—

1. Allow two or three drops of the gentian-violet stain to fall upon the slide and remain in contact with the film for *five seconds*.
2. Wash off the stain *with the iodine solution* applied from a drop bottle for five or six seconds. The film should then be black or dark brown.
3. Wash off the iodine solution with a mixture of 1 part acetone and 2 parts alcohol absolute, but allow to remain in contact for two or three seconds only.
4. Wash off with absolute alcohol, applied until no more stain comes away.
5. Wash in water, blot off superfluous water, and set aside to dry. If thought desirable, the preparation may be counter-stained by the application of a very weak solution of Ziehl-Neelsen.

Gram's method of staining has been used as a means of classification of organisms into two groups, those which stain (e.g., *B. anthracis*) and those which do not stain by this method (e.g., *B. typhosus*).

*Ziehl-Neelsen's method* is used chiefly for the bacilli of tubercle and leprosy, and for staining spores in bacilli. The carbol-fuchsin solution employed in this method is prepared by mixing

- 100 parts of a 5 per cent. solution of carbolic acid.
- 1 part of fuchsin.
- 10 parts of absolute alcohol.

The various stages of this process for staining a tissue section may be described as follows :—

1. Heat slowly the carbol-fuchsin solution (with the specimen in it) in a glass capsule on a sand-bath, in order to distribute the heat evenly. When the solution begins to give off steam, remove the capsule to the side of the bath, and allow the section to remain for five or ten minutes.

2. Remove the section from the carbol-fuchsin and place it in a capsule containing a 33 per cent. solution of sulphuric acid or of nitric acid, to decolourise it. In the acid the tissue loses its bright red colour and turns a pale yellow. Alternately remove it from the acid to the plain water and back again until it is of a *faint pink colour*.

3. Wash well to remove any of the superfluous stain.

4. Place in a capsule containing a saturated aqueous solution of methylene blue for one to two minutes.

5. Wash with water.

6. Pass through a 60 per cent. solution of alcohol in order to dehydrate.

7. Place in clove-oil to clear.

8. Mount in Canada balsam.

For staining by the Ziehl-Neelsen method an ordinary film proceed as follows :—

The film is prepared in the usual manner, either, for example, from the particulate matter of milk after centrifugalisation or from the lesions of an experimental animal, and fixed with alcohol and ether. Then stain as follows :—

1. Allow two or three drops of Ziehl's carbol-fuchsin to fall upon the film, and heat either over the flame or upon a warm stage.

2. Allow to act for three minutes, replacing with fresh stain any loss by evaporation.

3. Wash well in water, and treat with 25 per cent. sulphuric acid, or 33 per cent. nitric acid, until the film remains decolourised when washed with water.

4. Dry between layers of fine filter paper, and counter-stain with Löffler's alkaline blue.

5. Wash thoroughly, dry, and examine.

If milk is under examination it should first be centrifuged.\* The centrifuge tubes must be thoroughly cleaned

\* If no centrifuge is available the milk must be placed in a sterilised conical flask in the following manner: to 50 c.c. of milk set for sedimentation, 10 c.c. of liquefied carbolic acid crystals are added. The mixture is thoroughly

with water, followed by soaking for some minutes in strong sulphuric acid or nitric acid, then rinsed in tap water, distilled water, and finally rectified spirit. It is better not to place more than about an inch depth of milk in the tube. The centrifuge should be whirled for at least two minutes.

In the case of milk stained by this method, the bacilli of tubercle or other "acid-fast" organisms will be stained red, and the milk or casein cells blue.

It is important to recognise that the non-pathogenic "acid-fast" bacilli will be stained by this process as well as the pathogenic tubercle-bacillus. Some twenty different acid-fast organisms have now been isolated from milk, butter, grass, soil, dust, manure, etc. As this point is of great importance in differential diagnosis, it may be desirable to refer briefly to the subject of these "acid-fast" bacilli. The chief points for differential diagnosis between this group and the true tubercle bacillus are five:—(1) The tubercle bacillus shows a fairly uniform manner of growth; (2) it requires incubation temperature for growth in culture media; (3) it is unique with respect to its excessively slow growth; (4) it is as regards growth and propagation a parasite; and (5) on inoculation it produces pathological cellular changes distinct from the nodular new growths following inoculation of acid-fast bacilli. In particular this is true, as far as is known at present, in regard to the human organism. In a sentence, the acid-fast bacilli differ from the tubercle bacillus in three main particulars, viz.: morphology, conditions of development (chromogenicity, rapidity of growth, and wide range of temperature within which they flourish), and their feeble pathogenic properties. From these facts it follows that however great the degree of similarity between these various acid-fast bacilli, and however much it is possible by artificial cultivation to modify the morphology of the various forms, there is sufficient difference to enable a differential diagnosis to be made if all the biological characters are ascertained, and most of all the pathogenic properties. Hence the importance of the inoculation test being applied to all acid-fast and tubercle-like organisms detected in milk or butter. *The pathological differences* from Koch's bacillus are that inoculation with acid-fast bacilli gives rise to no "giant cells," no epithelioid cell clusters, and no tuberculous caseation. Nodular

shaken and poured into a tall conical glass. After standing for twenty-four hours a little of the sediment is taken by means of a sterilised pipette and the film made.

lesions occur suggestive of tubercle, but according to Potet, and Abbot and Gildersleve: (a) they constitute a localised lesion only, having no tendency to dissemination, metastasis, or progressive destruction of tissue by caseation; (b) they tend to terminate in suppuration like ordinary abscesses; (c) when occurring as result of intravenous inoculation they appear in the kidney, rarely in the lung and other organs; (d) the form of granuloma set up is similar to actinomyces. This group of organisms is one of considerable importance to the milk bacteriologist, and in all investigations dealing with the tubercle bacillus, or with milk and its products, it is essential that the acid-fast bacilli met with should be clearly differentiated from the tubercle bacillus. Not sufficient care has been taken in this respect up to the present. Any such organism found should be compared in cultural and pathogenic properties with the human tubercle bacillus, the bovine bacillus of pseudo-tuberculosis, and the various acid-fast organisms, and not simply accepted on tinctorial properties as tubercle bacillus.

**The Staining of Spores.**—The following are the methods commonly adopted:—

*Möller's method.*

(a) Prepare the film as usual, fix and dry, observing the precautions taken in preparing milk specimens.

(b) Treat with alcohol for two minutes, and then with chloroform for two minutes; wash in water.

(c) Treat with chromic acid, 5 per cent. aqueous solution, for from one to two minutes; wash and dry.

(d) Pour on freshly filtered carbol-fuchsin and warm gently till it steams; allow it to act for ten minutes and wash off with water.

(e) Decolourise with sulphuric acid (5 per cent.) and water alternately, to remove the carbol-fuchsin from the bacilli but not the spores.

(f) Dry and counter-stain with Löffler's blue until the film is of a faint bluish tint. Wash off stain, dry and examine. The spores will be stained red and the bacilli blue.

*Ziehl-Neelsen method.*

(a) Stain the film as for tubercle bacilli.

(b) Decolourise with 1 per cent. aqueous solution of sulphuric acid, or alcohol 2 parts, acetic acid 1 per cent., 1 part.

(c) Counter-stain with Löffler's blue.

(d) Wash, dry, and examine.

**Capsule Staining.**—*MacConkey's Method.*—1.5 gramme methyl-green crystals and .5 gramme dahlia are rubbed up in 100 c.c. distilled water: add 10 c.c. of saturated alcoholic solution of fuchsin, and make up to 200 c.c. with distilled water. Treat with stain for five minutes or longer, and wash *thoroughly* in a stream of water. The stain should be allowed to stand for a fortnight before use, and must be kept in a dark place.

By these or other methods of staining, specimens are prepared for the microscope. The microscopical examination yields information as to form, size, arrangement, spore formation, motility, etc.

## 2. CULTIVATION OR ARTIFICIAL MEDIA.

Various media, as soils for these microscopic plants to grow upon, are found to be necessary. Some of the commoner ones are broth, gelatine, agar, blood serum, milk, potato, and many special media for particular purposes. The property possessed by agar, which is prepared from Japanese seaweed, of remaining solid up to a temperature of 40° C., renders it specially useful for the cultivation of bacteria at temperatures between that degree and the melting point of gelatine (about 25° C.). There are many appliances which belong to a well-equipped laboratory which need not here be mentioned. It may, however, be desirable to state shortly what procedure is generally followed, in order to place the reader in a position to appreciate more fully much that has been mentioned in the text of this book.

A cultivation must be made on some selected medium in order to isolate one organism from another, and so obtain what is called "a pure culture" and also to secure valuable data in the way of characteristics of growth, in short, to allow the organism to exhibit its own biological character. To obtain pure cultures it is necessary to adopt Koch's *plate culture* method, or some modification thereof. This process is shortly as follows:—

A small quantity of the fluid or substance to be examined is mixed with ten or fifteen cubic centimetres of liquefied gelatine, which is then poured out into a flat Petri dish. The gelatine solidifies at a low temperature, and the plate is incubated at room temperature. In the course of a day or two, signs

of life appear in numerous "colonies," which represent individual bacteria, and are by this process of dilution separated from each other. It is now a simple process to examine these colonies by the naked eye and by the microscope to note their size, shape, configuration, consistence, colour, and arrangement in relation to each other, and whether or not they liquefy the gelatine. When these facts are ascertained, a sterilised platinum needle may be taken and colonies or parts of colonies removed and transplanted in agar, gelatine, milk, or upon potato or other medium, either as streak (or stroke) cultures, or as stab (or puncture) cultures.

*Stroke cultures* are similar in principle to plate cultures, and are made on the surface of a solid nutrient medium contained in a test tube, and which has been sterilised and allowed to cool into a solid state while the tube was placed in a slanting position, in order to afford a large inoculation surface.

*Stab cultures* are made in solid nutrient media contained in test tubes. The inoculation is effected with a sterilised platinum needle on the end of which a minute portion of the colony is taken up, and the needle is inserted for about an inch or more in depth into the nutrient medium along the middle axis of the tube, under the usual precautions against contamination. If the organism under examination is an ærobe, it will grow only on the surface of the medium; if an anærobe, only in the track of the needle; and if a facultative anærobe, it will grow both on the surface and in the track. We may obtain valuable diagnostic information from the presence or absence of liquefaction, gas-formation, rapidity of growth, or pigment production; and especially from the appearance of the colony in the track and on the surface. Moreover, when the organism is thus isolated in pure culture its further study is practicable.

The bacillus of *Diphtheria* grows very rapidly (12 to 24 hours), and appears as small scattered white colonies having a darker centre. Non-liquefying. The bacillus of *Anthrax* produces an arborescent growth of fine branching threads running out horizontally from the track of the needle. Liquefying gelatine. The *Cholera* bacillus has a characteristic mode of liquefaction. Koch's comma bacillus produces one large bubble at the point of inoculation which slowly increases in size. Finkler-Prior's bacillus liquefies rapidly and results in a tube of fluid with the organisms collected in a mass at the bottom. The streptothrix of *actinomyces* produces a dry, rough, raised, crinkled growth having a more or less bright yellow colour.



The bacillus of *Glanders* on glycerine agar has a uniform streak of grey-white colour of a shiny consistence. On potato it shows a characteristic growth of a brown-yellow tint on the third day and a reddish brown on the eighth. The *Tubercle* bacillus produces on glycerine agar in ten or fourteen days (it is one of the slowest growing of pathogenic germs) a raised, smooth, confluent growth which later becomes dry and irregularly crinkled. *Streptococcus bovis* appears in numberless, small, white colonies, non-liquefying and localised to the needle track. *Staphylococcus pyogenes* is a comparatively rapid liquefier and not unlike Finkler's Bacillus of Cholera. *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, *Micrococcus tetragenus*, and *Bac. pyocyaneus* are the four organisms commonly occurring in suppuration. The diplococcus of *Pneumonia* generally appears as an isolated and sharply defined line of growth with special luxuriance at the point of inoculation. The bacilli of *Tetanus*, *Malignant Œdema*, and *Symptomatic Anthrax* (Quarter Evil) are types of anærobic organisms, and the medium in which they grow is more or less split up by gas formation. The *Streptococcus* of *Erysipelas* grows almost exactly like the *Streptococcus* of suppuration. The organism of *Typhoid Fever* and *B. coli communis* do not show such marked features in cultivation as some other organisms, but they produce characteristic changes and possess biological features by which they are differentiated.

The bacillus of *Leprosy* has not yet been cultivated artificially.

Anærobes may be cultivated in an atmosphere of hydrogen or *in vacuo*; or various agents may be added to the media for removing the oxygen. There are a variety of methods for obtaining these conditions.

### 3. THE INOCULATION OF ANIMALS.

It is evident that to test the powers of disease-producing which any organism may possess, it is necessary to inoculate animals (*e.g.*, guinea-pig, rabbit, hare), to ascertain exactly what pathological effect is produced by the organism. A special licence is required in Great Britain for such experiments. The only common necessity in veterinary practice for such inoculation is in respect of the search for tubercle bacillus in milk. It will be sufficient to remark that the simplest forms of inoculation are all that are usually required in milk investigation, namely, the *intrapari-*

*oneal* and the *subcutaneous*. In some cases it may be sufficient to inoculate a few c.c. of the original milk; but, as a rule, it is advisable to centrifugalise, or use the sedimentation flask containing about 250 c.c. From the deposit or sediment two guinea-pigs may be inoculated, the one subcutaneously in the groin, the other intraperitoneally. Particularly is this necessary in making a reliable and exhaustive search for the tubercle bacillus. *Microscopic examination alone for this organism is not reliable*. The details of the process as carried out in practice are as follows:—After centrifugalisation the deposit is mixed with the 2 c.c. of milk remaining in the tube after aspiration of that which is superfluous. Two guinea-pigs (of say 250 grammes weight each) are taken and inoculated with the deposit from about 40 c.c. of milk. The fluid is inoculated subcutaneously on the inner side of the leg under strict aseptic precautions (the skin having been washed with 1-1000 corrosive sublimate, and shaved). In less than a fortnight's time, if the inoculated milk contained a considerable number of tubercle bacilli, typical infection of the popliteal and inguinal glands can be detected. If the milk contained very few bacilli the infection is much slower (fifth week). After the animal has been killed, the presence of the tubercle bacilli can be detected in the inguinal glands and the spleen. Some workers make it a rule to inoculate two guinea-pigs from the sediment of the milk, one receiving half of the sediment subcutaneously in the groin, the other receiving the remaining half intraperitoneally.

**Routine Procedure in the Bacteriological Examination of Cows' Milk.**—Physical examination (temperature, reaction, colour, cream, deposit, specific gravity, etc.) of the milk should be made if necessary. The microscopical examination of the milk before and after centrifugalisation or sedimentation will likewise often yield useful results.

1. *Plate cultivation*.—Dilute as required and make plate cultivations in Petri dishes. Six or more gelatine plates should be made and incubated at room temperature. Plates should also be made with nutrient agar for incubation at 37° C. Other media may also be selected. The plates should be counted on the second, third, and fourth days, and the necessary subcultures made. Agar plates incubated wholly at 18° or 22° C., will in the long run show more colonies than when incubated at 37° C. and then at 22° C., or at 37° C. throughout.

2. *Anaerobic cultivation*.—At the same time that the primary

aërobic plate cultivations are made, similar plates should be made on lactose-gelatine and lactose-agar for anærobic culture.

3. *Primary tube cultivation*.—Take ten tubes of 10 c.c. of the milk under examination and place three of them in the incubator at room temperature and three of them at 37° C. Place four of them in a water bath heated to 80° C. for fifteen minutes, and then enclose each of the four tubes in a Buchner's tube of pyrogallic solution or vacuum desiccator. These primary cultures may be tested in forty-eight hours for *B. coli*, the presence of indol, and *B. enteritidis sporogenes*.

4. *Secondary or subcultures*.—From the primary cultivations make subcultures on selected media for the isolation of organisms making their appearance on the plates, or, what is often preferable, make a separate set of plates for qualitative examination only.

5. *Examination for special micro-organisms*.—The milk must be centrifugalised or the particulate matter allowed to gravitate by sedimentation. It is, as a rule, useless to attempt examination microscopically or otherwise without first using the centrifuge or sedimentation flask. The deposit is then to be stained for the particular organism for which search is being made.

For centrifugalisation take two or three samples of the milk under examination to the amount of about 40 c.c. each and place it in the sterilised tubes of the centrifuge. In these tubes the milk may be centrifugalised for ten or fifteen minutes at 3,000 revolutions a minute. At the end of such a period the milk in each tube has separated into three layers—at the top there is a dense layer of cream, at the bottom there is the sediment or "slime" containing all the particulate matter, and between these two is the separated milk. Aspirate off the cream by means of a sterile glass tube connected with an aspirator or vacuum pump, and examine separately; aspirate all the separated milk except 2 c.c. The remaining sediment is so compact and dense that the tube may now be inclined and the sediment fully exposed without displacement. By means of a sterilised platinum loop a small portion may be taken up and spread on the surface of half a dozen slides and stained. The remainder of the sediment is well mixed with the 2 c.c. of milk and used for inoculation of guinea-pigs.

For sedimentation take two conical sedimentation glasses and fill them with the milk under examination, allowing them to stand in the refrigerator for twelve to fourteen hours. It is customary to add a few small carbolic crystals to each flask. On

the completion of sedimentation the milk has separated into three main strata, the cream at the top, the sediment at the apex of the flask, and the separated milk in the middle. The cream and milk may be decanted, and the sediment examined for any special micro-organisms.

6. *Inoculation of guinea-pigs* for the tubercle bacillus is the best available test for this organism. About 2 c.c. of the centrifuged milk is injected into the groin, and in two or three weeks local tuberculosis occurs if the milk contained the bacillus.

**Immunity.**—From what has already been said regarding the products of bacterial life, something of the basis upon which the great question of immunity rests will be understood. A culture planted upon an artificial medium will not continue to grow until it has exhausted that medium even under favourable conditions of temperature and moisture; for there comes a time sooner or later according to the particular species when the products of vitality oppose further growth. This occurs also in the body, and, broadly speaking, constitutes immunity. We know there is what is called *Natural Immunity*, a condition of the tissues or body cells which does not permit of certain diseases finding a nidus. Such a condition may also result as the sequela of the disease itself; thus we have an immunity partial or entire following small-pox or scarlet fever. This is termed *Acquired Immunity*. Recovery from any acute infective process under natural conditions is in principle the same. We know that we have at the outset of a disease certain poisonous substances produced which bring about the signs and symptoms of that specific disease. But as the disease progresses there are counter-substances produced which are antagonistic either to the poison itself or to the micro-organisms which have produced that poison. There is thus conflict within the body between the poisons or *toxins* and antidotes or *anti-toxins*. *Artificial Immunity* is nothing more than the artificial production of this result by imitating the natural process, that is to say, by inoculating some animal, preferably the horse, with the disease, in order to produce the toxins, and subsequently, therefore, the anti-toxins, in the animal's blood. If these anti-toxins are now extracted from the blood serum of the horse and inoculated into some animal suffering from the early stages of the disease, the same conflict will be set up in the second animal's tissues and fluids, and if the anti-toxins are sufficiently powerful to overcome the toxins which are being formed, the

result will be recovery or immunization, *viz.*, artificial immunity.

The question at once arises, What is it which makes the blood and body tissues antagonistic to the disease? The following are the chief theories which have been advanced to account for this condition.

1. *The Theory of Exhaustion*.—This now exploded idea supposed that there were in the body certain substances or conditions which were necessary or at least favourable to the existence of some particular micro-organism and its products, and that when that particular organism had once been present and had extended itself throughout the system, it used up these favourable substances or conditions to such an extent, that when the patient was again exposed to the infection, his body did not offer a favourable medium. Not only did this theory offer to explain the manner in which immunity was obtained from further attacks of a particular disease, but it was also supposed to account for the fact that the disease died out in the first instance; the pabulum being exhausted, the organisms succumbed and the patient recovered. Clearly, if this theory were true, the exhausted blood serum from such a case could not confer immunity or assist similar exhaustion in the blood serum of some other animal. But we know it can do this; therefore this theory evidently does not wholly explain the facts.

2. *The Theory of Retention*, which is the opposite of the foregoing supposition, assumes that the organisms produce substances which are inimical to their further growth. In a certain sense this theory probably is correct. It explains the limits of growth of any particular organism in an artificial medium in a test tube. But if it be applied alone to immunity in man it is obviously insufficient to cover the length of period of immunization.

3. *The Theory of Acclimatization* supposes an acquired tolerance on the part of the tissues to the toxic products of micro-organisms. "During a non-fatal attack of one of the specific diseases, the cellular elements implicated which do not succumb to the destructive influence of the poison acquire a tolerance to this poison which is transmissible to their progeny, and which is the reason of the exemption which the individual enjoys from future attacks of the same disease" (*Sternberg*).

4. *The Theory of Phagocytosis*.—Metchnikoff held that the introduction of micro-organisms into the living body caused an

inflammatory condition, the result of which was that a number of the cells of the body—leucocytes, endothelial cells, connective tissue corpuscles, etc.—took upon themselves the function of enclosing the intruding bacteria in their own protoplasm, and by assimilation destroying them; these scavenging cells he called *Phagocytes*. He held that the phagocytes were guided to the bacteria by a process of positive chemiotaxis. The theory has met with considerable opposition, as an explanation of immunity, and yet contains an explanation of certain ascertained facts.

5. *The Antitoxin Theory*.—Roux and others hold that these substances, which may for the moment be allowed to pass under the somewhat generic term of *antitoxins*, are produced *in the body cells* as a result of the interaction of the living body cells with the toxins. The anti-toxins are held in solution in the blood, and exert their influence in opposition to the toxins. Ogata observed that anthrax blood contained some substances which neutralised the products of the anthrax bacillus. Behring, Kitasato, and others added very much to our knowledge of these products of organismal life by their investigations into Tetanus and Diphtheria. These may be called at their outset *local* diseases, in which the site of the specific bacilli is a strictly localised one; yet it is well-known how general the disease almost immediately becomes. Dr. Sidney Martin has well summed up the position as follows:—"Microbes produce by their growth and multiplication in the tissues certain toxins which, while present in the blood and tissues, furnish these with power to inhibit and kill the same species of micro-organism." And again: "As long as these antitoxic substances remain in the tissues the body remains insusceptible."

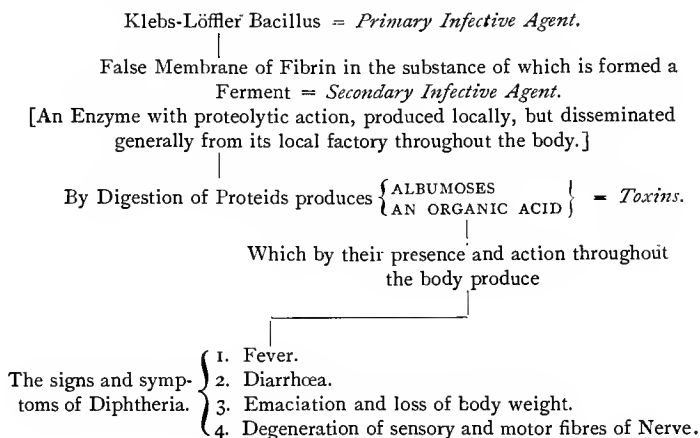
Recently Ehrlich has suggested a theory of explanation which has been called the *side-chain theory*. He regards a molecule of protoplasm as composed of a central cell group with a number of side chains, by which the central or mother cell receives nutrition from outside its own economy. Such side chains have therefore been called *receptors*. They are of two kinds, first those having a single unsatisfied combining group, attracting and absorbing molecules of simpler constitution; and secondly, those having two such groups, one for food molecules and the other for fixing ferments in the fluid medium around. Now when toxins are introduced into the system they are fixed, like food molecules, by the receptors, and the receptors

are lost and shed off into the blood firmly combined with the toxin molecule as an inert body. This is repeated as further toxin is forthcoming and thus arises an hypertrophy of the receptors which then occur in increased numbers in the blood, not combined with toxin cells, but as free receptors constituting antitoxin molecules.

The exact hypothesis accepted is of less importance than the recognition of the broad fact that toxins in the blood and tissues *stimulate a production of anti-toxic bodies in the body cells* arising as far as is known from the body cells, and these anti-toxic cells protect the individual from the injurious effects of the toxins with which they unite, producing inert compounds resulting in immunity.

As an illustration of the practical working of the antitoxin theory, we may consider the position in Diphtheria as far as the somewhat confusing nomenclature in vogue will permit us to understand it. Diphtheria is a severe inflammation and fibrinous infiltration of the mucous membrane of the fauces, larynx, and trachea, leading to a necrosis of the superficial part of the mucous membrane, which it thereby changes into a tenacious, greyish-white, pseudo-membrane. This membrane is not necessarily limited to the throat, but may occur in the stomach, intestine, external generative organs or wounds. The membrane consists of an infiltration of the submucosa with round cells and fibrine. The bacilli of Klebs-Löffler are held to be responsible for the production of the pseudo-membrane on the surface of the mucous membrane, and the bacilli are found, with many others (streptococci, etc.), in the superficial and deep layers of the membrane. The bacilli exude a poison which is of the nature of a proteolytic ferment. This ferment is absorbed, and by digesting the proteids of the body, chiefly those of the spleen, produces *albumoses* and an *organic acid*, which are the toxins or specific agents in producing the signs and symptoms which clinically constitute the disease known as Diphtheria (*Martin*). The albumoses are always associated with an organic acid, and are much greater in amount than the acid. Both acid and albumoses are what are called Nerve Poisons, and bring about the degeneration of nerves, etc., which characterise Diphtheria. The albumoses, however, when injected into rabbits, produce a high temperature and diarrhoea as well as paralysis, which affects first the posterior limbs, afterwards the respiratory muscles, and finally the heart. The changes in the nerves, according to Martin, are

well marked. The axis cylinders break up into segments, and the medullary sheaths become ultimately affected. Fatty degeneration takes place in the muscle fibres. The organic acid has practically the same action as the albumoses, but in a less degree. Now these changes and the theories explaining them may be graphically represented in a sort of scheme :—



Such a table is of course a crude and provisional representation. But broadly it states the view generally accepted. The same sort of plan could be made in *Tetanus*, in which we have a disease affecting chiefly horses and man. A wound becomes contaminated by the primary infective agent, Nicolaier's *Bacillus tetani*, which almost invariably remains in that locality and does not pass into the circulation. Martin, Brieger, Kitasato, and others have isolated toxins which have a fever-producing action or cause muscular spasms, and here, as in Diphtheria, it is likely that a ferment is concerned in the toxin production, which toxin, also like Diphtheria, leaves the site of its birth and passes throughout the body, especially affecting the spinal cord.

We are now in a position to consider the practical formation of antitoxins—naturally and artificially. As we have already seen, bacteria produce disease by two main factors : first, by the multiplication of the living organisms in the body to which they have gained entrance ; and, secondly, by the production of toxins. In the course of time these toxins are either transformed by the body cells, or, by their interaction with the body cells, by a process at present unknown, are able to produce



antagonistic substances which are inimical to their own action.

To obtain these substances artificially for inoculation in cases of disease, a virulent culture of the disease is inoculated into an animal, whose blood serum, in the course of time, will produce antitoxins. It is then drawn off, and used for inoculation as a protective or defensive agent. We shall best follow the details of the process by taking a concrete example, which shall be Tetanus :—

1. *The Preparation of the Toxin.*—Grow a pure culture of Tetanus in hydrogen in broth, and allow it to grow thus for four weeks, after which, filter through a Chamberland filter. After this  $1\frac{1}{10}$  ccm. ought to kill a guinea-pig (say of 250-300 grammes), and it is by estimating its lethal power in a standard guinea-pig that the strength of its toxicity is determined.

2. *Immunization of Horse with Toxin.*\*—The toxin is generally treated before inoculated into the horse by the addition of Gram's iodine solution in the proportion of 1 : 3.† Injection is made subcutaneously in gradually increasing doses. This process continues through some eight weeks, until the horse is able to stand without injury a very considerable dose of toxin.

3. *Preparation of Antitoxin.*—This of course is carried on in the blood and living tissues of the horse, and the blood serum is from time to time tested by withdrawing a small quantity and observing its power against an exact amount of toxin. When the required degree of antitoxic power is reached, the animal has a three or four days' rest, and blood is then drawn off under aseptic precaution from the jugular. It is allowed to coagulate, and the serum separates. This is collected, and contains the antitoxin, which is now ready for use, and may be kept (preferably with a small percentage of carbolic acid or other preservative agent added) in solution or, by evaporation over acid *in vacuo*, in crystallised form. Serum yields roughly 10 per cent. dried substances, which should be kept in a cool, dark place.

4. *Clinical Therapeutics.*—It is clear that the sooner the antitoxic serum is injected into the patient suffering from Tetanus, the better. The results of Diphtheria compare

\* The horse is used on account of its size and susceptibility to tetanus.

† Terchloride of iodine may be employed also; and other methods of attenuation have been tried with success.

favourably with Tetanus in this respect for the simple reason that in the former it is possible to diagnose the condition comparatively early, whereas, in Tetanus the condition cannot be detected till in an advanced stage. The disease in the patient has, so to speak, *to be caught up*. It has had the start. The immunity conferred by the injection of antitoxin is but temporary, lasting only a few weeks; but clearly that is sufficient to tide over the crisis. More efficient methods, more careful and exact dosage, the adoption of Dr. Cartwright Wood's double toxin method, and an earlier application, are almost certain to give increasingly favourable results. In Tetanus, Professor Kanthack pointed out that serum treatment has proved of little avail in acute cases in man with a short incubative period, but in chronic cases with a longer incubation, the results have been more favourable. Much the same process of production of toxin and antitoxin for therapeutic use is followed in Diphtheria.

Before closing these scattered remarks upon immunity it may be useful to add for reference Hankin's nomenclature. Buchner proposed the term *alexins* (defenders), and Hankin subdivided these bodies into *sozins* and *phylaxins*. The *sozins* are defensive proteids which are present in the normal animal; the *phylaxins*, on the other hand, are the defensive proteids which are present in the animal after it has been made immune artificially. It is these bodies to which the term *antitoxins* would be generally applied in the narrow sense of the word. Hankin further subdivided *sozins* and *phylaxins*, accordingly as they acted on the bacillus or its toxins, into *myco-sozins* and *toxo-sozins*, and *myco-phylaxins* and *toxo-phylaxins*.

## CHAPTER VI.

## THE TERMS INFECTION AND CONTAGIUM.

BY M. H. HAYES, F.R.C.V.S.

SINCE ancient times, many attempts have been made to classify certain diseases which, respectively, have a more or less uniform type, and which are set up in the animal body by causes that cannot be perceived by the unaided senses of man. They have been divided, according to their respective modes of occurrence, into *epizootic*, *enzootic*, and *sporadic diseases*, which divisions are neither distinctive enough nor sufficiently well defined to merit adoption. Some of the diseases in question were found to be communicable from one animal to another by direct or mediate contact, and were consequently said to be "*contagious*" (Lat. *contagio* = contact); while others, such as bovine pleuro-pneumonia and rinderpest, having demonstrated their ability to go from one host to another independently of contact, were termed "*infectious*." This attempted classification was mainly one of degree; for the large majority of the so-called infectious diseases (such as foot and mouth disease and variola) are also contagious. As instances of the few purely contagious diseases, I may mention rabies and dourine. Besides, the distinction, as regards the mode of transference, between a so-called contagious disease and a so-called infectious disease is often more apparent than real; for, as cases in point, the dried sputum of tuberculosis, or the dried bacilli of tetanus, may, on being carried by the air in the form of dust, set up their respective diseases in a manner which might appear to an unscientific observer as "*infectious*"; although the transference of the virus is as mechanical as that by the lancet or inoculation syringe. With advance of knowledge, so many of these so-called contagious and infectious diseases were proved to be caused by special micro-organisms, that pathologists felt justified in including among them a large number of similarly behaving diseases (such as distemper and variola), the exciting causes of which remain still unknown. As these microbic and supposed-to-be-microbic diseases had

the common property of possessing a contagium, or virus, by means of which all these diseases had been proved to be, or were supposed to be, propagated, whether from outside or from one animal to another, a classification founded on this property seemed to be as reasonable as it was convenient. The special meaning attached to the word "virulent" precluded the use of the adjective derived from "virus." The necessity of avoiding ambiguity prevented their being termed "infectious diseases," than which "contagious diseases" was a still more objectionable designation. The not very happy escape out of this difficulty has been to give them the name of "*infective diseases*." An infective disease, taking for granted its microbic origin, may be defined as one that is caused by a living micro-organism which is capable of becoming developed in the animal body.

Although the condition of minuteness is purely arbitrary, it is well recognised in practice: for example, we withhold the term "infective" from mange, which is set up by an acarus; but we concede it to surra, which is caused by an infusorian. The tendency to become general is in most cases considered to be a characteristic of infective diseases; parasitic ring-worm, which is due to a microscopic mould, being classed as a skin disease.

The words "*volatile*" and "*fixed*" are employed with reference to the capacity the contagium or virus has or has not of being carried to a distance from its place of development, whether such place is outside or inside the animal body, and independently of the mode of disease transference from one animal to another. Thus the virus of that non-contagious disease, South African Horse Sickness, is fixed; and that of malarial fever, which is a non-infectious disease, is volatile.

When the exciting cause of a disease can, like that of anthrax or tetanus, exist independently of the animal body, we apply the expression *ectogenous* to it. The cause of an *endogenous* disease, on the other hand, is one which, like that of glanders, can fulfil its life mission only in its animal host. Hence, in the case of an endogenous disease, the destruction of all infected animals, with the allowance of a reasonable length of time for the death of the special organisms, would prevent its further occurrence; but such action would have no such result with an ectogenous disease. A *miasmatic disease* is an ectogenous disease, the contagium of which is volatile.

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